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VETERINARY MEDICINE

OCTOBER 1938

CONTENTS

Saliva Testing of Race Horses....430
A Good Hoof Oil....433
The Horse in South Africa....434
Equine Intravenous Medication....436
Restraint for Treatment of Hind Feet....441
Some Feedlot Diseases of Lambs....442
Pregnancy Disease of Ewes....445
General Purpose Ointment....445
Infectious Necrotic Enteritis....446
Ecto-Parasites and Mange Mites of Swine....450
The Swine Industry....453
Useful Prescriptions for Swine....454
Plant Poisoning in Swine....456
Coöperation Between Veterinarians and S. P. C. A....460
Treatment of Septic Conditions of the Phalanges....463
Drugs Used in Veterinary Medicine....464
Dysentery of Sheep....469
Canine Pediatrics....470
Poisonous Plants of the South....477
Abstracts of Recent Veterinary Literature....478-480
Infectious and Metabolic Diseases, 478; Chronic Catarrh in Horses, 478; Roaring in Horses, 478; Causes of Sterility in Cattle, 478; Purpura Hemorrhagica, 479; Electro-Thermal Treatment in Horses, 479; Cystic Calculi in a Mare, 479; Summer Sores in a Horse, 479; Shipping Diseases of Cattle, 480; Vitamin E Deficiency in Cattle, 480; Causes of Retained Placentae, 480.

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In general the feeding of irradiated yeast has no significant effect upon the resistance of the udder to the invasion of mastitis streptococci.—Tech. Bul. No. 243, N. Y. State Agric. Exp. Sta.

Good quality, high yielding cheddar cheese cannot be made from milk produced by cows infected with active mastitis.

Bovine mastitis streptococci were found to survive in the experimental low curd tension milk cheeses after nine months.

It is important that mastitis be eliminated as far as possible from herds supplying milk for cheese making.—Tech. Bul. No. 242, N. Y. State Agric. Exp. Sta.

Vitamin D fluid milks have been on the market for about six years. They now have come to be favorably regarded by most of the leaders in the field of nutrition, public health and medicine. This has been brought about in part by clinical studies which have defined the relative values of the different types. Of late, wider recognition is being given to the fact that growing children, as well as infants, need a regular intake of vitamin D, and the potential field of usefulness for vitamin D milks has thus become enlarged. The outlook for the future is promising.—*Vitamin D Milk.*

Mastitis, because of its usual insidious nature, seldom receives the attention deserved on account of its extent and the serious economic loss it entails. Udders, apparently normal in every respect, on physical examination oftentimes harbor the mastitis organism. . . . Critical study of the extent of mastitis in a given number of dairy cows, including tests conducted for the purpose of making as accurate a survey as possible, revealed the presence of this disease in numerous quarters of udders in which no abnormality of any kind could be detected upon physical examination, and in which the owner had not detected any clinical manifestations of the disease.

It is estimated that the present consumption of vitamin D milk has reached a volume of about 400,000,000 quarts annually. It is encouraging to note that, even though, for the country as a whole, only 3 per cent of all fluid-milk consumed is vitamin D milk, in some centers of population it constitutes as much as 6, 9 or even 20 per cent. However, the present volume, large as it is of itself, represents but a small fraction of that which must be attained if vitamin D milk is to make its maximum possible contribution to the advancement of public health.—*Vitamin D Milk.*

Saliva Testing of Race Horses

By L. L. NEWMAN, Pullman, Washington

SINCE its beginning about four years ago, the saliva testing program has taken rapid steps toward its development and ultimate perfection.

Previous to 1932 eight states permitted legalized betting on horse races; now approximately 30 states permit betting through legislative action. Along with the reëstablishment of legalized betting came all the so-called evils of racing. The one in which I have become especially interested is the doping menace.

In American racing the idea has become generally accepted that aged, rheumatic, and unsound thoroughbreds require a stimulant before going to the post. This is not necessarily to increase their speed but to compensate for their infirmities. The dangers which arise from such a practice are evident.

Under the Harrison Anti-narcotic Act unauthorized possession of narcotics is a felony and the federal narcotic agents started investigations. Their activities led to every track of prominence in New York, Maryland, Illinois, Michigan, Ohio, Florida, Louisiana and Kentucky. As these investigations continued, the federal narcotic agents found that the practice of doping horses had assumed sinister proportions. This led to widespread humanitarian outbursts.

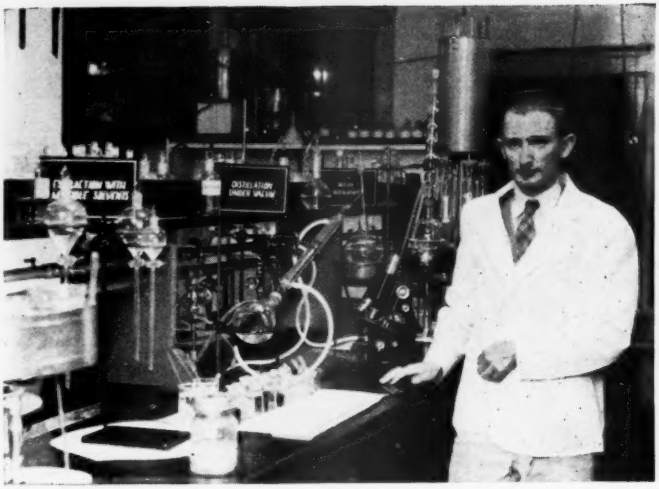
Some of the stables raided were equipped like pharmacies with apothecary scales, weights, measuring glasses, test tubes and an assortment of drugs such as heroin, cocaine, codeine, strychnine and digitalis. Scores of prescriptions for drugging horses were seized. One prescription confiscated called for the following: 1½ grains heroin, ¼ grain strychnine, 2 drops nitoglycerin, 5 drops digitalis, 2 ounces kola nut, with directions to give half the amount 50 minutes before post time, and the other half 20 minutes before post time.

Legalized betting having been reëstablished in Washington, it was the Commission's desire to maintain it as a permanent and respectable institution. This could be done only by thoroughly destroying its bad aspects. With this in mind the saliva testing problem was presented by the Washington Horse Racing Commission in the fall of 1933 to Dr. E. E. Wegner, Dean of the College of Veterinary Medicine of the State College of Washington. At this time active interest was taken by the Department. Previously little work had been done on doping in the United States; in fact, the first work was done in 1933 by Dr. J. G. Catlett, veterinarian, and Dr. Charles E. Morgan, chemist, who were appointed by the Florida Racing Commission to work out a program of taking saliva and analyzing it.

O. B. McRory, Ph.C., an undergraduate of the College of Veterinary Medicine, and the writer, a senior, took up the problem. It was presented to us in embryo. The only material available was that of the procedures followed in France. Drs. Catlett and Morgan had not at this time progressed sufficiently in their work to materially aid us. With the aid of Dr. N. G. Covington, pharmacologist and physiologist at the State College of Washington, our experiments were conducted in the veterinary College until the spring of 1934, when racing opened at Longacres race track at Seattle. The College of Veterinary Medicine and the Washington Horse Racing Commission have given us their utmost coöperation and best facilities for continuing the saliva testing program.

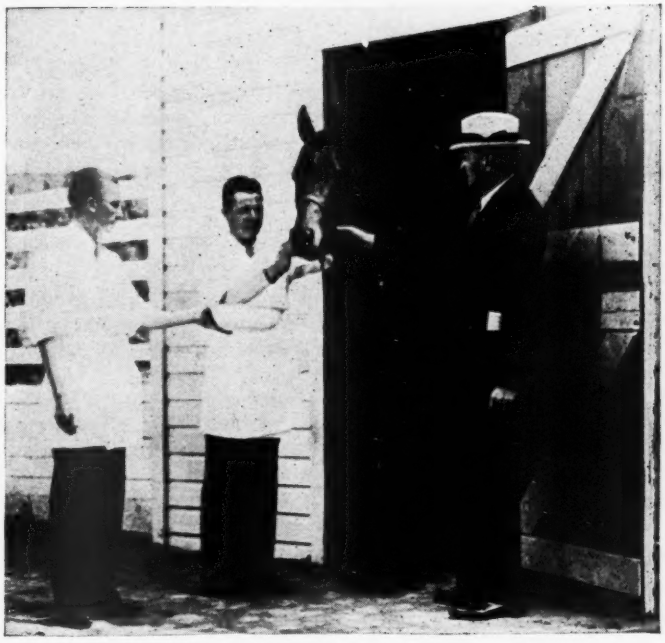
During the past four years, saliva testing has attained its high status through the excellent coöperation of the different men working upon this problem, and it is through their efforts that saliva testing was adopted by the National Racing Association.

The State Racing Commission employs a veterinarian to represent it. It is his duty to take the saliva sample and prepare it for immediate shipment to the designated saliva testing laboratory which, in our state, has been the State College of Washington. The efficiency with which the test is taken has a great deal to do with the results. It is im-



Left: Dr. O. B. McRory in his testing laboratory at the State College of Washington.

Below: from left to right: O. B. McRory, L. L. Newman and the late A. R. Galbraith. Doctor Galbraith was a member of the Washington State Racing Commission, Doctor Newman and Doctor McRory were employed by the Commission to collect and analyze saliva specimens.



perative that several cubic centimeters of saliva be collected, sealed, and preserved properly. This must be done in the presence of witnesses and in a scientific manner which can be upheld in any court. Every precaution is taken to prevent contamination of the saliva after the owner, trainer or his employé has been notified to present his horse for the saliva test. It is our policy to have an assistant employed by the Commission accompany the horse from the time of notification until the taking, sealing, and acknowledgment of the proceedings have been completed.

When the horse has been presented to me for the taking of the saliva, I take from an envelope a pair of sterile cotton gloves and put them on in the presence of the person accompanying the horse. Then from another envelope I remove three sterile gauze tampons, made from 100-yard rolls of Rutger's 8-ply dressing gauze. These are cut about 12 inches long and folded three times. Two of these tampons are placed in an enamel pan. Then the seal is broken on a 2-ounce screw cap bottle containing sterile distilled water which is poured over the tampons in a collecting receptacle. The third

tampon is kept dry. The old saying "Gain the animal's confidence" is a notable suggestion. The temperament of these horses is often exceedingly erratic. One false pass and you may have a little trouble, especially since the horse has just completed a strenuous race. Twitches, tongs or ear holds many times do more harm than good. The horseman's suggestion should be considered. I approach the horse from in front on his right side, the horseman standing on the left. I take hold of the bridle ring or chin strap or put my hand on the horse's nose. This type of approach gives a person a chance to protect himself, observe the eye, and smell the breath. My assistant holds the collecting basin under the lips. Holding one of the moist tampons with a heavy 12-inch straight dressing forceps, I swab the mouth, cheeks and teeth. The horse may or may not object to this; usually quietness will render the procedure easy and the animal will like the feel of the water in its mouth. It is allowed to chew the gauze, as this helps stimulate the flow of saliva. Massage from the outside over the Stensen's duct also aids the flow of saliva. The second tampon is used in the same manner as the first. The tongue is now grasped by the left hand and pulled out, and the cheeks, teeth and tongue are dried with the third (dry) tampon. Each tampon after being used is placed in the basin with the saliva which has dripped from the horse's lips.

After the sample is taken, the horseman witnesses the preserving and sealing of the sample. A pint jar of the Ideal Ball type is used. This jar, sent to me sterilized and sealed by the chemist, contains 60cc of 95% alcohol. The seal is broken, and the contents of the basin and the gloves are placed in the jar and sealed with a sticker which bears the sample number and date. This, in turn, is sealed with a lead wire seal. A representative witnesses the cutting at random into two parts of a 2½-inch by 1-inch label; the line is always cut zig-zag. Both halves of this label contain the registration number of the saliva. The left half is moistened and placed on the acknowledgment and stamped with a seal of the Commission. The right half is then torn off and placed on the sample jar; this gives indisputable identification when the chemist returns this label with his results.

The horseman signs an acknowledgment in duplicate which includes the complete procedure: name of the horse, owner, trainer, jockey, distance of race, date, race number, track and who took the test. The originals are turned in to the Commission each evening after the races.

Having worked two years upon the extraction of alkaloids from saliva, I will give a sketch of the analysis. It is by no means a veterinarian's job to analyze these samples unless he has the proper training. In court a veterinarian's word against a chemist's would amount to little. The analysis consists of a series of extractions with suitable solvents, such as chloroform or ether, under conditions either basic or acid to pick out the different alkaloids. After having the extracts designated as A, B, C, they are evaporated. One knows what alkaloids may appear in the extract. One may try for a general chemical reaction to see if any alkaloids are present; then, if there are, specific chemical and microchemical tests are made. As we all know, some reagents produce a more delicate reaction than others. We as veterinarians in sending in these samples must remember that this is dealing with new toxicological processes where very minute residues are obtained. It is my understanding that toxicologists will not give a positive report until they can identify the drug by three chemical reactions.

A person doping a horse has one of two things in mind; either it is to stimulate or to depress the animal. The degree of stimulation or depression will depend upon the drug or combination of drugs used. He must also consider the timing of the drug's action and plan his method of administration, which may be any one of those we are familiar with, including the rectal method. Hypodermic injections are given in areas where they will not be noticed, such as in the tip of the ears, under the mandible or in the chest region. Often by this method the point of injection is not discernible. The timing must be absolutely accurate in order to be efficient. If the desired amount of re-

action from the drug does not take place at the right time, it is valueless; so, on many occasions the horse has either run his race in his stall before the race, in the paddock, or in the stall after the race.

In addition to timing we have to consider the concealing of the "hop" drugs with other drugs or by mechanical means, either directly in the mouth or included in the prescription. Drugs put in the mouth can often be smelled or their effects seen in the mouth. We have found atropine in samples of saliva when the mouth before moistening with the tampon was dry as a bone. Urine or blood samples may be taken if the amount of saliva taken has not been satisfactory. Horses usually urinate when being cooled out or shortly after. It is an easy matter to make the collection then. For one season I took urine samples, but rely now entirely upon the saliva for analysis unless for definite reasons a urine sample is desired. It is true that some drugs are more easily detected in the urine, but they are also eliminated in the saliva which takes much less time to collect.

The clinical symptoms are not as well defined today as they were a few years ago because of the horseman's fear of being detected. The amount or degree of stimulation is very small. We do not see horses climbing fences with their eyes showing the characteristic symptoms of doping. One of the best symptoms I find is this: I feel the horse's pulse and watch the trainer.

No doubt new problems will be presented to the veterinarians and chemists yearly; however, at present the status of saliva testing is such that we are able to cope with doping in such a manner to rid the "sport of kings" of its most deadly enemy.

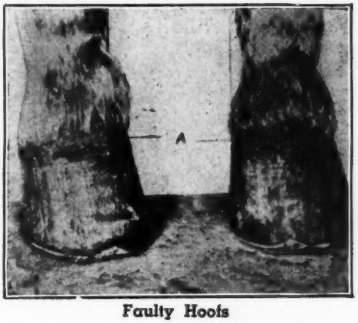
* * * *

A practitioner should perfect himself in the various methods of administering medicines or biologic products to horses. Nothing makes such a good impression on the owner or attendants as skillful handling of this important subject. To be able to administer a capsule, hypodermic or intravenous solution to the animal with the least possible resistance on the part of the patient is the first step in equine practice.

A Good Hoof Oil

A preparation we have used for years on founder, thrush, gravel, contracted heels and almost every kind of hoof trouble is the following, which we call "red oil":

In a large mortar of at least half a gallon capacity, cut two ounces of gum camphor in small pieces. Pour one ounce of commercial sulphuric acid over this, and



Faulty Hoofs

stir with a glass rod until it is a deep brown color.

Pour in one quart of turpentine, stirring constantly with the glass rod. The solution will get hot and smoke considerably.

In several hours, stir in two ounces of oil eucalyptus with red oil color. Stir well and allow to stand overnight.

The next day, with a dose syringe, siphon off the clear oil without disturbing the sediment. The latter, mixed with light mineral oil, makes a good hoof dressing.

This red oil can be used undiluted, or mixed with various oils.

Some years ago we accidentally let the door on the rear deck of our coupé down on our right thumb. The pain was intense, and we stuck the injured member in a wide-mouthed bottle of this oil carried in the car. To our surprise, the pain soon stopped, and the only complication remaining was a slightly thickened nail, which became normal in a few months.—*E. T. B.*

* * * *

The client who pays his bill is the client who is satisfied.

* * * *

Some horses die getting well, while others recover that should, by all the visible symptoms, succumb.

The Horse in South Africa

By G. Martinaglia, M.Sc., D.V.Sc.,
Johannesburg, South Africa

THE horse is not indigenous to South Africa. In 1654 van Riebeek, the founder of the Cape, wrote to East India for horses and received in response animals from Java, which were of poor quality. In 1689 Persian stallions were introduced and in 1778 South American sires were imported. The first English blood stock arrived in 1792.

Although a hardy animal, many authorities consider that the South African or Cape horse is poor in bone and would gradually degenerate, but for the introduction of fresh blood from the British Isles. The South African Government, fully aware of this, offers every encouragement to importers of pedigreed stock.

The South African horse is lighter than the North American animal and, therefore, not so well suited to plowing and other farm work, which is done, for the most part, by oxen. The horse thus plays a less important rôle on a South African farm than in the United States or Canada.

IN South Africa, private horse practice is undertaken by a small but able body of veterinarians only in the cities. The field of practice comprises racing stables, polo and hunt clubs, riding schools and privately-owned studs. Rarely is the city veterinarian called to a farm as the Government Veterinary Department offers free service to the farming community. Progressive municipalities employ a staff of veterinarians who are responsible for the health of their equine transport animals, so that they, too, fall without the scope of a private practitioner.

The horse, like the ox, is closely bound with the early history of South Africa and both have played an important part in the development of the country. With the help of his noble steed, the pioneer was able to carry the torch of civilization northwards, and history abounds with tales of perilous journeys and of record rides, proving the stamina and endurance of the Cape horse. As an example there is the stirring story of Woltemade, who rode seven times to a sinking ship and saved fourteen lives. Finally both horse and rider were drowned. There is also the story of Dick King's famous ride when he sought help for the garrison at Durban, six hundred miles from Grahamstown.

THE romantic coach days saw numerous horse or mule teams operating between the coastal towns and the gold and diamond fields of the interior. During that period many important business transactions were dependent on equine speed and endurance. All that has passed with the advent of the automobile, the electrification of street cars and the mechanization of Government, municipal and private services. Inventions of the 20th century have brought about a gradual decline of the horse as a utility animal. This has been evident not only in the cities, but also on the farms. Devastating diseases, too, have been responsible, to some extent, for this gradual decrease in number.

AFRICAN horse-sickness and biliary fever (piroplasmosis) have proved a menace particularly to the farmer. After long summer rains horse-sickness often occurs in virulent cycles and may leave certain areas almost denuded of horses. Sir Arnold Theiler devoted many years to the study of this disease and prepared a serum, administered intravenously in large quantities, which in the case of mules proved effective against the malady, but which in the case of horses was often accompanied by serious untoward results, such as acute liver atrophy and tendo-vaginitis.

Nieschulz demonstrated that horse-sickness can be conveyed to mice by intracerebral injection of the virus. By further experiments, Alexander and du Toit, of Onderstepoort Government Laboratory, near Pretoria, have been able to produce an attenuated neurotropic virus vaccine from mice brains, which is used extensively. It is cheaper and more easily applied than the

old method of immunization with serum, and there are no complicating sequelæ.

This important discovery has given the farmer fresh hope for the future of the horse and may prove to be an important factor in rehabilitating horse-breeding in South Africa.

Biliary fever, due to its tick-borne nature, is easily controlled in the cities, but presents a difficult problem in the country. Let us hope that the day is not far distant when this disease will be as effectively treated as piroplasmosis in dogs.

Dourine, another disease of horses, has recently assumed serious proportions in the Cape Province. The Government Veterinary Department is carrying on a vigorous campaign against dourine, but the many adverse factors, e.g., unfenced farms, in this country do not promise a speedy eradication as in Canada, where its suppression must be acknowledged as one of the great achievements of modern veterinary science.

APART from the communicable diseases already mentioned, other serious equine maladies are of rare occurrence. In cities, sporadic diseases and lameness are common. Animals in the Johannesburg municipal services are more prone to lameness than to any other complaint, so that the Municipal Sewerage Farm, cultivated with Italian rye grass, has proved beneficial to patients sent there to recuperate.

The death rate from disease in Johannesburg is exceedingly low and some mules have a record of 21 years in the municipal service. The greatest mortality is due to that curse of modern civilization—street accidents.

The South African farmer loves his horse; the Basuto native is proud of his hardy mountain pony; and racing will always be a popular sport for the city dweller. It would seem, therefore, that in this country there is hope for the future of the horse. As a draft animal he may be ousted from the streets, but he has no equal as a useful companion of man, and he is unsurpassed for beauty and symmetry. Urban and rural admirers will insure his perpetuation.

MANUEL H. SARVIDE, MEXICO, D. F.

Entered the National Veterinary School (Mexico) in 1923.

Assistant Professor of Histology National Veterinary School 1926-1929 inclusive. Obtained professional degree Veterinary Doctor granted by National Autonome University of Mexico, 1930; thesis dealing with cancerous histology.

Professor of Histology and Elements of Embryology National Veterinary School 1930—. Professor of Pathological Anatomy and Histology since 1934. Professor of Biology 1931-1935. Professor on Genetics at the National Agriculture School, 1933-1936. Chief of the National Veterinary School Laboratory 1933-1935, and Director of the School since 1935. Technical Tuberculinization Inspector of the Public Health Department of Mexico.

Fred M. Hayes Succumbs to Heart Attack

Fred Montreville Hayes died at his home in Davis, California, June 12, 1938, from a heart attack.

Doctor Hayes was born in 1885 in Barnesville, Kansas, and was graduated from the Veterinary Division of Kansas State College in 1908. He taught at his Alma Mater until 1911, when he left Kansas for the College of Agriculture, University of California, where he rose to the position of Professor of Veterinary Medicine.

Going to California when veterinary service in the state was new he had much to do with bringing about its present excellent condition. His activities covered many phases and he has many excellent publications to his credit in the fields of hog cholera and Brucellosis. Doctor Hayes has been President of the California State Veterinary Association and on important committees of the A. V. M. A. and the U. S. Live Stock Sanitary Association. He was also a member of many scientific societies.

Equine Intravenous Medication

By EUGENE B. INGMAND, Zionsville, Indiana,
Allied Laboratories, Inc.,
Pitman-Moore Biological Laboratories

THE advantages of intravenous medication are obvious. In the first place, introduction of therapeutic agents directly into the blood stream permits a rapid distribution and quick absorption by the most intimate cellular structures. This time factor is exceedingly important because by the time the veterinarian sees the patient many cases have reached the stage when life and death are separated by only a few minutes.

The importance of the time factor is likewise apparent in certain virus diseases. All are aware of the generally unsatisfactory attempts to treat active cases of hog cholera and our more successful results in treating other virus diseases, in which the serum can be administered promptly by the intravenous route. In hog cholera the intravenous injection of anti-hog cholera serum is out of the question, but in infectious equine encephalomyelitis, for example, the intravenous injection of serum is a common procedure. Even granting that there are certain immunological differences in the character of these viruses, the fact is: a potent serum, when given intravenously, is able to ferret out the virus and exert its full therapeutic effect promptly, whereas if this serum be given subcutaneously, valuable time is lost in absorbing it.

Recent work along this line has revealed that once a virus attaches itself firmly to a tissue cell, no amount of serum, regardless of its potency, is able to dislodge it. Thus, it is apparent that the quick intravenous route is the most suitable in such cases.

Another advantage is that certain drugs, which if given subcutaneously or intramuscularly would produce swelling and even abscesses at the site of injection, may be administered intravenously with no local reaction. Such drugs include solutions of calcium chloride, calcium gluconate, concentrated solutions of dextrose, and epinephrine.

Equipment

The amount of the product to be injected will determine to a large extent the equipment necessary. In the case of small doses, 40cc or less, the average serum syringe is very satisfactory. For injecting larger quantities of such fluids as calcium gluconate, physiological salt solution, dextrose solution and blood, it is necessary to use a large container, which provides for the delivery of the solution to be injected either by gravity or under pressure. Fortunately, the commercial containers of products intended for intravenous use are easily adapted to the various intravenous outfits on the market.

One outfit consists of a 6-inch air vent needle which is thrust through the rubber stopper until the end is above the liquid when the container is inverted. A short 15 to 16 gauge 1½-inch needle is also inserted through the rubber stopper. To this is attached a 5-foot length of rubber tubing. An adapter is attached to the other end of the tubing. Then an intravenous needle (2 to 3 inch, 15 to 16 gauge) is inserted into the vein. The container is then inverted and the free end of the tubing, containing the adapter, is attached to the needle in the vein. By raising or lowering the bottle, the rate of flow can be controlled. If pressure is desired, a rubber bulb may be attached to the air vent needle.

A more recent development is the "simplex" intravenous outfit, which operates by means of gravity and is exceedingly practical. The advantage of the outfit is that it is all rubber (it contains a non-metal flutter valve) and will fit 250 to 500cc containers. It is attached by removing the rubber stopper of the container and slipping the hood, which contains the flutter valve, over the neck of the valve. To this valve is attached a convenient length of rubber tubing, a needle adapter and an intravenous needle.

It is highly important that all equipment

used for this type of work must be not only clean, but sterile. In fact, the veterinarian should conduct these intravenous injections in the same manner as he would any other operative procedure. He must be clean, his apparatus must be sterile, and the product must be delivered into the blood stream in the proper manner.

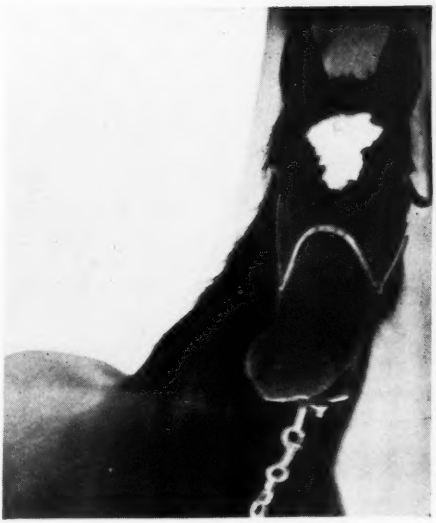
Sites and Technic

In the horse the jugular vein is the most suitable site. It is readily accessible and large enough that injections can be made with ease.

The injection of drugs and biologics via the blood stream should be carried out in a careful, aseptic manner. The site of injection should be clipped with scissors and shaved in so far as possible. The area should then be disinfected with a suitable antiseptic.

Having prepared the injection site and made ready the equipment and product to be injected, the vein is distended and the needle thrust through the skin into the vein. When the needle is in the vein, it should be extended as far as the length of the needle will allow along the lumen of the vessel, as this will tend to prevent its coming out during the course of the injection. The syringe or delivery tube is connected to the needle. It is important that the connecting tube, which runs from the bottle to the needle inserted in the vein, be filled with the fluid, so that when it is connected to the needle there is no danger of air passing directly into the blood stream. While the dangers of air emboli have perhaps been over-emphasized, it is needless to allow such errors to occur.

The flow of fluid from a syringe or from a container under pressure should be very slow at first. A rate of 10 to 15cc per minute for the first minute should be a rule. Then the speed of flow may be increased, this increase depending to a certain extent upon the reaction of the animal to the product. For example, the administration of products such as calcium gluconate in 250 to 300cc doses should consume at least ten minutes. Too rapid injection of such products causes them to reach the heart in a too concentrated form, often causing shock,



Too Inquisitive

Apparently this horse stuck his nose into some insect's business. No traumatism of any kind could be found. With applications of white lotion and belladonna, the swelling subsided in several days.

which may be termed speed-shock. It is not necessarily due to the character of the product, but rather to the overtaxing of the heart by a substance foreign to the organ. This question of time is important to the veterinarian because if too much time is consumed in making the injections, they are uneconomic. However, if the veterinarian is to make intravenous injections properly, he must administer these products in a careful manner and the time factor cannot be overlooked.

Another point of practical interest is the temperature of the agents injected. Under ordinary circumstances pharmaceutical agents for intravenous use should be at about body temperature. However, a range of 10° to 20° below that temperature is usually not objectionable. The thing to remember is that the product should not be *above* body temperature. Immune sera may be given at body temperature, or they may be chilled without producing any untoward reaction.

We may now consider some of the products for intravenous use and their indications.

Dextrose

Dextrose, as a therapeutic agent, has a number of uses in the practice of veterinary medicine. Basically, this agent is a food representing the end product of carbohydrate metabolism; it is actually the only form in which foods of this nature can be utilized without modification by the animal body. From the standpoint of medicine it is apparent that a product which can be assimilated immediately upon administration has a distinct advantage over foods which require digestion. Animals which cannot take food per os, can be nourished by the intravenous injection of 5 to 10% dextrose. Five per cent dextrose in physiological salt solution or distilled water, which is commonly used for this purpose, is isotonic with the blood and therefore will not exert any deleterious influence.

In regard to the diluent for dextrose, either distilled water or physiological salt solution may be used. There is this difference; if the salt solution is used, the fluid portion of the solution injected will not be eliminated as rapidly as when distilled water is used. This should be taken into consideration when dextrose is used intravenously over a period of several days, because of the edema which may develop if the salt solution alone is employed. A practical way to handle this problem is to use the dextrose in a salt solution for two days, then in distilled water for the next two days, continuing to alternate the two solutions at two-day intervals.

Dextrose in a 50% solution is a splendid diuretic, and in certain instances may be used for flushing the urinary tract. This diuretic action of 50% dextrose may likewise be used to advantage in treatment of forage poisoning. The value of dextrose in the treatment of shock is well established. Dextrose in a final concentration of 10% in physiological salt solution is also a product of value in combating dehydration and supplying nutrition.

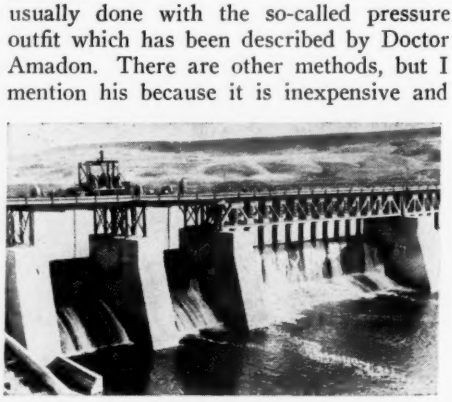
Physiological Salt Solution

Physiological salt solution has several uses in the field of veterinary medicine. Its principal use is in replacing lost fluids, with respect to volume. This loss of fluid may occur as a result either of hemorrhage or of shock. The caution regarding physiological salt solution is repeated: its continued use will result in the development of edema. Frequently acacia, in a 6% concentration, with 0.6-0.9% physiological salt solution as a diluent, is used for the purpose of restoring lost blood volume.

Another condition in which salt solution should be used even more generally is that commonly called heat stroke. Strychnine, camphor and oil, and cold water enemas are used in an effort to reduce the temperature and support the heart, but all the symptoms manifested in cases of this nature are probably due in the main to loss of sodium chloride. While the drugs mentioned constitute good therapeutics, the injection of sodium chloride intravenously is what the animal actually needs. Here we are treating the cause of the disease, not the symptoms.

Blood

Blood is not a cure-all, nor will it be used every day in veterinary practice, but it certainly has advantages and is almost specific in the treatment of certain diseases. The first problem here is the collection of the blood. The best way is to collect it in a suitable container which contains sodium citrate as an anticoagulant. After the amount of blood that is needed is collected, it is injected into the animal to be treated. This is usually done with the so-called pressure outfit which has been described by Doctor Amadon. There are other methods, but I mention his because it is inexpensive and



Irrigation Dam
Huge irrigation dams in the West mean the establishment of thousands of small farms, largely powered by horses or mules. Veterinarians are interested.

convenient for a general practitioner. The anticoagulant, sodium citrate, can be purchased as a specially prepared, liquid solution for this purpose. The final concentration of this chemical in the blood should be at least 0.3%; it may be well to increase this concentration to 0.6%, because of the variable conditions under which the blood is collected upon the farm. The citrated blood is injected intravenously into the patient under pressure or by gravity. The use of the pressure outfit, which is very similar to that used in injecting calcium gluconate, enables one to regulate the flow of blood. This is important because at the start of the injection the blood should be given slowly and then, according to the reaction of the patient, the rate of injection may be increased or decreased as required.

Uses of Blood

In conducting blood transfusions by indirect method there is always the problem of incompatibility of the bloods of the donor and the recipient. It is therefore important to determine whether the serum of the recipient will agglutinate the blood cells of the donor. A test to determine this point consists in mixing a small amount of serum of the recipient with a few drops of the saline suspension of cells of the donor. If the blood of the recipient is incompatible with that of the donor, there is a marked clumping of the cells in a manner similar to that of a positive rapid plate agglutination test for Bang's disease.

In cases of navel ill, blood is generally beneficial; doses of 250 to 350cc are usually sufficient. Debilitated animals, especially those which are extremely dehydrated and refuse to eat, are greatly stimulated by these injections. Blood is likewise indicated in shock, especially where the traumatic shock results from the accumulation of blood in the capillary bed, and in cases of hemorrhage due to accidents and following surgery.

Anesthetics

It is hardly necessary to mention the use of nembutal intravenously because its value to the veterinarian is known to all. In the horse chloral hydrate may be used intravenously with very satisfactory results. It must be used in a relatively dilute solution (7-10%); must be administered very slowly, and it is contraindicated in debilitated animals. In such cases there is no difficulty in producing anesthesia, but the subjects many times fail to rally from the effects of the drug. The anesthetic dose varies from one to two ounces, depending upon the susceptibility of the animal to this drug. The period of complete anesthesia varies from one-half to one hour, with a recovery period of one-half to one hour, during which the animal regains consciousness and is able to stand.

Arsenical Preparations

Two other drugs which may be used to advantage intravenously are sodium cacodylate and neoarsphenamine. Both of these agents may be used in the treatment of equine influenza and pneumonia. They are convenient to administer because of the small bulk, are quickly disseminated to all parts of the body, and are able to exert their full therapeutic effects accordingly. Sodium cacodylate is used more generally because it is cheaper than neoarsphenamine, but in cases where it is possible to use neoarsphenamine, it is the agent of choice. The dose of sodium cacodylate is 45-90gm and it may be repeated at 24-48 hour intervals. In the case of neoarsphenamine the adult equine dose is 3 to 4.5gm.

Hydrochloric Acid

Hydrochloric acid is commonly used in a 1-1500 solution. Its value is based upon its effect on the blood and tissue colloids, stimulating leucocytosis and phagocytosis, and thus increasing body resistance against bacterial infection. Hydrochloric acid has a wide variety of uses where bacterial pathogens are the cause of the disease. For example, in cases of pulmonary infections of the influenzal variety, this type of therapy is frequently beneficial. It may be alternated with sodium cacodylate or neoarsphenamine in the treatment of equine pneumonia and influenza.

Immune Sera

In using highly potent sera therapeutically it is necessary to inject such agents in a manner which will allow dissemination of

the protective substance throughout the body as quickly as possible. The intravenous route is admirably suited for this purpose, and all initial injections of immune sera for therapeutical purposes should be given intravenously.

Dyes

A group of therapeutic agents worthy of mention is the dyes. While dyes have been used to a certain extent, their application in veterinary practice is in its infancy. Though Erlich's dream of one dose sterilization of the blood stream has never been realized, the value of such therapy should certainly not be overlooked in veterinary practice. Acriflavine, a flavine dye developed by Erlich, should probably be considered more seriously by the veterinarian. In a dilution of 1:1000 it is not toxic, and is indicated in cases of septic metritis, blood stream infections, pyemia and other bacterial infections. The dose should be at least 100cc and may be repeated at 24 hour intervals.

Speaking of Horses
Here is Horsetail Falls along the Columbia River Highway, near Portland, Oregon.

Purgatives

Lang's solution, a combination of sodium chloride and sodium citrate, is a relatively new product and sufficient time has not elapsed to determine its true virtue in the treatment of cecal impaction and constipation. The suggested dose is 500cc and should be administered very slowly to prevent overtaxing the heart. It is contraindicated in "heavey" horses.

Dr. S. L. Stewart recommends the use of eserine salicylate ½gr and pilocarpine hydrochloride 2gr (for 1200-1600-lb. horses) dissolved in 250cc sterile distilled water and administered by the intravenous route. The solution is indicated in cases of acute and chronic indigestion and particularly in cases where the toxic products are beginning to act on the vital nerve centers.

Azoturia

Several intravenous products have been used in the treatment of this condition: calcium gluconate, 250cc; calcium chloride, 100-250cc, 20% solution; and a 10% solution of sodium sulphate in a dosage of 100cc. While these products have not produced consistent results, they are worthy of consideration.

Hemostasis

While sodium citrate is extensively used as an anticoagulant, it appears that in large doses it is a very valuable coagulant. The dosage suggested is 30gm, dissolved in 90cc of warm distilled water. It should be injected slowly. A solution of calcium chloride should be handy in case of shock. This solution of sodium citrate is indicated in preventing operative and post operative hemorrhage, and surface and nasal hemorrhage.

Restraint for Treatment of Hind Feet

In my practice I have found many cases in which it is necessary to manipulate the hind foot of a horse. It is difficult to hold some animals when it is necessary to clean out a nail puncture, or to do other work in which the sensitive laminae are entered.

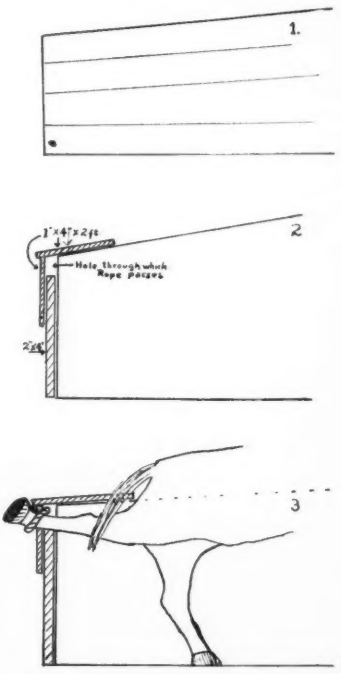
During my first two years in practice, we usually cast these animals, especially if we were unable to find a husky farmer to hold the foot; many lost their strength when such a job was at hand.

When treating as many as six of such cases in a single day, casting became too laborious. We therefore adopted the following method, which we have used ever since.

The left side of a straight stall is used if the operator is right-handed; at the end of this a three-foot two-by-four is spiked three to four inches short of the top of the stall. A one-by-four piece about two feet long is nailed over and beyond the two-by-four, leaving an opening through which the rope is passed. A similar one-by-four piece is nailed on the top. The end of the two-by-four holds the rope up; the extra small pieces keep it from sliding off, and prevent the horse from kicking over the top.

The horse is tied short to the front of the stall with a strong rope, his breast up to the manger. The rope is tied with a quick-release slip knot. The horse is twitched, and the owner holds the twitch, with instructions to pull the knot free in case the animal falls. A rope is also placed at the side of the horse to keep it against the left side of the stall.

We carry a five-eighth inch rope about 30 feet long for such work and for raising down horses. One end of the rope is tied around the fetlock of the affected foot (either right or left) with a quick-release slip knot. The other end is then passed through the hole in the end of the stall into the next stall around a post or through a hole in the manger. My assistant holds this rope and takes up the slack as the foot is raised. In this manner the foot is brought to the edge of the stall, held secure, and the necessary work can be accomplished easily with the protection of



1. Side of stall; 2, showing pieces of timber nailed to the stall, and 3, position in which the foot is held.

the stall between the operator and the animal.

This may seem a lengthy procedure, but we have not found it to be so. It may also seem a little dangerous for the animal, but during the four years we have used this method, we have never had an accident. Some animals kick and fight, and a few throw themselves or jump into the manger, but in such cases, we simply repeat the procedure, and we always accomplish the job.

It is wise to leave the stall so arranged, for the owner is more likely to take the proper aftercare of the foot. He usually calls the veterinarian, because he is unable to hold the foot.

W. V. Stevens.

Holgate, Ohio.

Some Feedlot Diseases of Lambs*

FRANK THORP, JR.,
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The Author

DISEASES among lambs in feedlots may be divided into two general groups, those resulting from shipping and those due to poor feeding practices.

Overeating

This difficulty probably accounts for greater losses among Colorado feed yard sheep than all other lamb diseases combined. The condition has been called "apoplexy", "indigestion", "gastroenteritis", "food intoxication", "overfeeding" *et cetera,* but the significant cause is undoubtedly the eating of too much concentrated food.

The feeds causing the most trouble in this state are corn, barley and peas. Ensilage prepared from corn grown on irrigated land, often contains far more grain than is realized and may therefore contribute to the disease. It is likely that starchy feeds, such as the pulp, crowns and tailings of sugar beets, when fed in conjunction with grain may enhance this condition. Such feeding practices as allowing lambs to harvest corn and peas directly from the fields have been discontinued as it is impossible to control the amount of feed consumed.

There seems to be no question but that overfeeding of the aforementioned grains is the exciting factor, but it is impossible to state that a given amount of grain will kill lambs. In one feed yard the sheep may be consuming twice the amount of grain consumed in another yard, while the greater losses may be occurring in the latter feed lot.

Research workers in England, Australia and Tasmania have shown that braxy-like diseases are associated with a toxin developed in the intestinal tract of sheep by a microorganism of the *Cl. welchii* group. At the Colorado Experiment Station, a toxin in the intestinal filtrates prepared from lambs dying of overeating has been demonstrated. This finding has not been constant, however.

The condition of overeating invariably affects the largest, fattest, greediest and most vigorous lambs in the feedlot. Often the animals are found dead, without anything abnormal having been observed. The symptoms may include: (1) holding the head back; (2) walking with a jerky gait; (3) animals becoming excited and attempting to run, stagger and fall, continuing to move all four legs; (4) vomiting of grain in some cases; (5) ceasing to eat; (6) diarrhea lasting for several days; and (7) rapid loss of weight. Death follows shortly except for an occasional recovery. In some cases the lambs become temporarily paralyzed and are unable to stand for weeks. Pneumonia is often a complicating factor in recumbent cases and frequently hastens death.

The most constant lesion encountered in overeating is inflammation of the intestines and congestion of Peyer's patches. Inflammation of the abomasum may occur. In some acute cases no lesions may be observed; in other acute cases large, blotchy hemorrhages may be found under the peritoneum over the large and small intestines, diaphragm and abdominal wall. Frequently petechiae are observed under the epicardium and on the thymus gland. A straw-colored exudate, which may or may not be coagulated, is often found in the pericardial sac. In acute cases sugar is invariably present in the urine but is absent in chronic cases.

Overeating is fairly easily diagnosed when fat lambs die with no evidence of pneumonia and when bacteriological findings are negative. The presence of sugar in the urine of acute cases is regarded as

*Contribution from the Colorado Agricultural Experiment Station, Fort Collins, Colorado.

diagnostic, as is also excessive quantities of grain in the rumen and reticulum. Undigested grain is frequently found in the abomasum, small intestines and cecum. Overeating is often erroneously diagnosed as hemorrhagic septicemia.

The condition invariably disappears in a few days if concentrated food is decreased in amount or omitted from the ration. Since concentrated food is the predisposing factor, control would seem easy; since after the removal of grain from the ration losses stop very quickly. However, the difference between a fattening ration and a killing ration is slight and the problem of control entails practical difficulties. Care is required to bring the lambs to full feed without causing digestive disturbance. Measures that prevent gorging should be instituted. The lambs should be sorted into groups of as nearly equal size and weight as possible, the small animals being fed less grain than the larger ones. The grain should be evenly spread in the troughs and no more trough space allowed than is necessary to accommodate the lambs. In fact, the animals should stand so close together at feeding time that if one should leave the feed line there would be no possibility of finding another opening.

By feeding a variety of foods, overeating is satisfactorily eliminated. Ensilage, beet pulp and molasses should be included in the grain ration, and less grain fed. The lambs should be scrutinized carefully, at each feeding time, to see that none are off feed and that the fat lambs are not getting more than their share. Scouring should be regarded as a warning sign.

Coccidiosis

Coccidiosis develops within the first two to three weeks after the animals arrive in the feed yard and therefore may be classed as a shipping disease. The cause of coccidiosis is a protozoan parasite (*Eimeria faurei*) which multiplies in the mucous membrane of the large and small intestines. The coccidia are ingested with contaminated food and water.

—*Courtesy B. A. I.*

Corriedale Ewes, United States Sheep Experiment Station, Dubois, Idaho.

The most perplexing factor in coccidiosis is the finding these parasites in the feces of lambs within one day after unloading from the train. The factors involved in an outbreak are difficult to explain. If lambs arrive carrying coccidia, what causes the parasites to become virulent? If infection is dependent upon the number of coccidia ingested, it would seem that shipping and penning the lambs would always furnish the necessary conditions for an outbreak but this does not always occur.

The affected animals become dull and lethargic, lose their appetities and suffer from diarrhea. The discharges soon become bloody. The diarrhea may continue for several days causing the lambs to become weak, lose flesh and die. The disease may spread rapidly and cause many deaths or it may be chronic and affect only a few animals. Enteritis may be present in both the small and large intestines. In the cecum gray streaks are often present and in the small intestines gray, blotchy areas usually visible to the naked eye

through the peritoneal coat. The disease is easily and positively diagnosed by microscopic examination of the feces. When it exists the coccidia can be found in great numbers by direct examination of the matted wool around the anus.

Affected animals should be isolated. Exposed but healthy individuals should be allowed to run over a large acreage to lessen reinfection. The flock should be examined daily and the lambs that are scouring removed. If this is not possible the pens should be cleaned every two to three days. Feeding native hay, oats and bran seems to be beneficial.

Soremouth (contagious ecthyma)

This disease is caused by a filterable virus and usually appears during the first week or ten days after shipping. The disease is, in itself, not very serious, but if complications develop losses may be heavy. The *Actinomyces necrophorus* is a secondary invader in this malady and often a serious complication.

Soremouth is characterized by the appearance of small vesicles on the lips, gums and sometimes on the tongue. As the disease progresses the lesions become crusted with a brownish scab; at which stage the lips are likely to be swollen and to appear thickened. Such animals lose their appetites and may lose flesh. If swollen scabby lips are observed in certain members of the flock a critical examination will often reveal a mild infection of the lips of almost every lamb.

When the disease is confined to the mouth few losses occur, but when lesions appear in other parts of the body the condition may become serious. The following complications have been observed: (1) laryngeal lesions simulating calf diphtheria; (2) ulcers in the rumen, reticulum, omasum, abomasum and small and large intestines; (3) small necrotic foci in the liver, and (4) pneumonia characterized by circular necrotic areas.

Vaccination of young lambs is the most effective means of preventing the disease. The vaccination of a flock of 700 lambs showing 10% infection on the second day after arrival, was followed with good results. No further cases developed and the affected animals exhibited no further development of lesions during the two months they were under observation.

Hemorrhagic Septicemia

This condition usually appears shortly after the lambs are received and rarely occurs after the third or fourth week. The disease is caused by the microorganism *Pasteurella oviseptica*. The conditions that render the animals susceptible are not entirely understood but it appears that shipping with subsequent chilling, driving and hunger are the predisposing causes. During shipping, the animals may also contact carriers of virulent microorganisms.

By the time the disease is noticed a few animals may be dead of the acute form, while other members of the flock are dull and show mucopurulent discharge from the nose and a watery discharge from the eyes. A cough develops, the appetite is diminished, breathing rapid and temperature elevated. Animals dying of the acute form of the disease have hemorrhages under the pleura along the ribs, on the heart and on the thymus gland. An excess of straw-colored fluid is frequently found in the pleural cavity and within the pericardium. Congestion of the mucous membrane of the trachea and bronchi is often apparent. Congestion and hemorrhages may occur in the mucous membrane of the abomasum and duodenum. In the less acute cases hemorrhages are less frequent and pneumonia is the outstanding lesion.

Under good conditions of housing, weather and feeding, the disease is self-limiting. Isolation of sick animals should be practiced. The value of vaccination during an outbreak seems questionable in view of experimental work with cattle. If vaccination is carried out it should be accomplished two to three weeks before shipping.

The aforementioned diseases are by no means all that affect feeder lambs. They are, however, those of greatest economic importance. In the diagnosis of these diseases a complete history is important as is also a pathological and bacteriological study in addition to the clinical examination.

Pregnancy Disease of Ewes

In cases unable to stand, and carrying twins or triplets, the mortality in our practice has been around 95%. Only young and extremely hardy ewes seem to survive. If treatment is given while the animals are yet able to stand or walk, the loss is around 40%. If treatment is given when the first clinical symptoms are noted, about 85% can be saved.

The symptoms, while somewhat variable, usually begin with a lack of appetite. The ears have a peculiar "clammy feel" when palpated, much like that of a cow coming down with milk fever. Constipation, probably due to a complete stasis of the gastric and peristaltic action, is present. Later on, the animal acts dizzy, blind, and finally loses control of the hind legs.

Pregnancy Disease

In small flocks of valuable sheep, when one or two cases have occurred, much trouble may be prevented by giving all the others a prophylactic treatment.

Treatment consists in giving 50 to 125cc of calcium gluconate with dextrose in the axillary space. Ground oats, molasses, iodized salt, good hay, sufficient water with the chill taken off, abundant exercise and a dry bedding place, are all important hygienic measures. An easy way to give molasses is to put a quart of molasses in a ten quart sprinkling can, and then fill the can up with medium warm water. This can be applied to either grain or hay.

In serious cases, add 5cc sterile camphorated oil, with a tenth grain of atropine to the gluconate solution.

Autopsies

The importance of postmortem examinations in sheep practice cannot be over emphasized. An autopsy may be the means of holding a client under the most adverse circumstances. Let the owner view the "innards" and he usually will be satisfied. Furthermore, you will learn much from it.

General Purpose Ointment

An ointment that can be used almost every day in practice, is composed of the following:

Adeps	lb. 10
Petrolatum	lb. 5
Cera flava	oz. 4
Pix liquida	oz. 16
Phenol	oz. 4
Liq. cresolis com.	oz. 8
Ol. cajuput	oz. 8
Ol. sassafras	oz. 2
Kerosene	oz. 8
Sulphur sublimatum	lb. 2

Melt the lard, vaseline and wax in different containers, and mix well in a large dishpan.

Have the liquid ingredients warm. First add the tar and stir well. Then add the other liquid ingredients and keep on stirring. Lastly, add the sulphur, which has been sifted, with more stirring. As it cools stir frequently. A smooth, tan-colored, neutral smelling ointment results.

It is useful for itch, footrot, wire cuts, abscesses, skin troubles, etc. in horses, cattle, sheep and hogs. It relieves irritation; repels flies; freshens up old sores, and is useful for summer itch in horses. It is a mainstay in everyday work. It works so well that it discourages home made mixtures of creolin, sulphur and "fryings."

Stored in ten-pound lard pails, it will hold up in the hottest weather. Straight adeps has a tendency to "run" in warm weather, but the other ingredients hold this in check.

Infectious Necrotic Enteritis

By A. T. KINSLEY,
Kansas City, Missouri

Infectious necrotic enteritis of swine is widespread and prevalent. It is one of the factors responsible for post-vaccination losses.

Extensive losses in stock hogs, previously immunized against hog cholera in public markets, have been due to infectious necrotic enteritis. Infectious necrotic enteritis should not be confused with so-called bloody dysentery, coccidiosis, spirochetosis, trichomoniasis or balantidiasis.

Occurrence

Pigs weighing 30 to 50 pounds are most frequently affected, although this infection may occur in recently farrowed pigs and in aged hogs. Breed appears to play no part as regards susceptibility to this disease. The physical vigor of the swine, undoubtedly, is a factor in susceptibility; however, shotes seemingly in the best of condition become affected. Climatic conditions have little or no influence in the prevalence of infectious necrotic enteritis, however, the disease appears to be more common in the late spring and early autumn, probably because that is the season when the major portion of the pig crop is at the most susceptible age. Sanitary conditions in which the swine are maintained have an influence upon the susceptibility of swine to the infection; nevertheless, the disease may prevail in swine that are kept in apparently clean sanitary quarters. Food seems to have little, if any, influence upon the prevalence of the disease, although insufficient or poor quality of feed possibly predisposes to this as to other diseases. It occurs in garbage fed hogs and in swine fed a grain ration.

Etiology

From the experimental data available it seems justifiable to state that the majority of the cases of infectious necrotic enteritis in swine is primarily caused by *Salmonella suipestifer*. Other microbian agents such as *S. enteritidis* and *S. aertrycke*, may be the primary cause of some cases of enteritis and either one or both may occur as complications in *S. suipestifer* infections. Of 50 outbreaks of infectious necrotic enteritis *S. suipestifer* was isolated in 40; *S. enteritidis* in nine and *S. aertrycke* in one outbreak. The *Actinomyces necrophorus* may be demonstrable in the lesions of enteritis but probably has no primary etiologic relationship. However, this microbe as a secondary invader may assume a leading rôle after the field has been prepared for its growth. No doubt, intestinal parasites are important predisposing factors because of the injury they inflict on the intestinal mucosa, thus providing a favorable field for infection.

Contaminated feed or water taken is the usual source of the infection.

Lesions

The principal lesion of infectious necrotic enteritis involves the intestinal mucosa. The acute type of the disease is typified by congestion and inflammation of varying intensity. The spleen may or may not be tumefied.

The subacute and chronic forms of this disease are characterized by subacute and chronic inflammation respectively of the intestinal mucosa. In some cases the lesions indicate that the infection begins in the lymphoid tissue (Peyer's patches and solitary follicles) of the intestinal mucosa near the ileo-cecal valve. The lesions may be found in any portion of the digestive tract including the stomach, but are usually more intense in the intestine.

In the beginning the lymphoid areas become congested and tumefied; later they undergo necrosis. In other cases the mucosa becomes congested, tumefied and necrotic. When large areas of the mucous membrane become involved, the lesion appears as a diphtheritic inflammation because of the adherent necrotic tissue. In some cases the entire intestinal wall appears thickened and friable, due to the accumulated necrotic tissue. Some cases are observed in which the necrotic tissue

has sloughed, leaving the intestinal wall relatively thin and inelastic.

In prolonged cases or in the chronic form of this disease the so-called "button ulcer" is of common occurrence. This lesion occurs when the infection is localized in solitary follicles or Peyer's patches and the inflammatory and necrotic processes extend by continuity, the necrotic tissue remaining intact or accumulating as a dirty brown mass with raised margins. Microscopically the surface epithelium in the affected areas and the lymphoid tissue are found to be in various stages of degeneration and disintegration. The degenerated and dead cells commingle with the coagulated inflammatory exudate and appear upon the surface as an adherent mass—the "button".

The mesenteric lymph glands, through which the lymph passes from the infected mucosa are tumefied and may be congested and hemorrhagic. In the subacute and chronic cases the central portion of the lymph glands may be necrotic. The peritoneum, adjacent to the involved intestine, is invariably affected with a subacute or chronic inflammation. The peritoneal inflammation is manifested in the early stages by an excessive outpouring of serous fluid, but as the process continues there is a tendency toward organization and limited fibrous proliferation, which is prone to produce adhesions.

Symptoms

The symptoms of infectious necrotic enteritis are variable. The intensity manifested is not proportional to the intensity and extent of the lesions. Hogs apparently in the best of health may, on autopsy, reveal lesions indicative of a chronic infectious enteritis, while swine manifesting the usual symptoms of this disease may show very slight lesions on autopsy. The infection acts rapidly and is highly destructive in some cases but is slow-going and rarely fatal in other cases.

An acute septicemic type of *S. suipestifer* infection occurs, although rarely. It is characterized by a high temperature, inappetence, arched back, frequent urination and "blue belly."

The subacute and chronic types of this disease may affect only a few animals in a herd. Again few animals may be affected at one time, and they either die or recover; followed by the infection of others and a repetition of the process. Thus, the disease ultimately affects all swine on a given farm. In other instances practically all of the swine show evidence of the disease at the same time.

The affected animals seem dull; they separate themselves from the other swine.

The appetite is diminished, although this may not be noticed by the caretaker. In some outbreaks the first evidence of the disease noted is the general dullness. The owners will report that a large percentage of the hogs lack activity and appear dull and depressed. As the disease progresses, digestive derangements become more evident and are manifested by irregular appetite and diarrhea. At this stage, the animals appear unthrifty, have rough coats, and arched backs. The fecal discharges are quite fluid and of variable color, depending upon the feed.

The affected individuals become emaciated, thin and weak and may die of exhaustion or they may ultimately recover. The temperature varies from normal to 105° F. or 106° F. during the active stage of the disease, but in the later stages it is not unusual to record subnormal temperatures.

Pneumonia is occasionally associated with enteritis and is apparently caused by the *S. suipestifer*. The pneumonic lesion is similar to the lesion that is associated with infestation with larval ascarids except that the ascarid pneumonia is metastatic and

the *S. suipestifer* pneumonia is of an atypical lobar type.

Diagnosis

The characteristic lesion upon which judgment is based occurs in the intestine and consists of an inflammation in which some of the involved tissue has become necrotic and is adherent to the intestinal mucosa. The finding of these lesions when coupled with a definite history of the outbreak and observation of the leading symptoms, irregular appetite, diarrhea, dullness and variable temperature, is usually sufficient evidence upon which to make a positive diagnosis.

A common condition observed on many farms. The swine covered with mud indicate a hog wallow is maintained on the premises. The small pig on the extreme left is a runt. Runts are due, usually, to parasites. Hog wallows, ascarids and runty pigs are commonly found together.

Differential Diagnosis

Subacute or chronic infectious necrotic enteritis is manifested by unthriftiness, rough coat, diarrhea, a temperature up to 105° F., and an irregular appetite. Enteritis with a diphtheritic exudate of varying intensity involving the large intestine is the characteristic lesion of this disease.

Acute Gastro-Enteritis is characterized by an acute inflammation of the intestinal mucosa, the large intestine being primarily involved. The spleen may be extensively enlarged. There may be a bluish discoloration of the skin and nephritis.

Swine dysentery is usually signalized by a relatively sudden onset. There is marked depression and a bloody diarrhea. The lesions consist of an intense hemorrhagic enteritis and usually sloughing of varying sized areas of the involved mucous membrane. The affected intestinal mucosa is usually brick-red in color with no organized inflammatory exudate.

Erysipelas in the acute form begins with a high temperature, inappetence and frequently also retching and vomiting. The lesions are parenchymatous degeneration of vital organs, edematous tumefaction of lymph nodes, hemorrhages and inflammation of the gastro-intestinal mucosa, the small intestine being primarily involved. The serous coat of the stomach may show the so-called "paint brush hemorrhages."

Coccidiosis is manifested by unthriftiness and evacuation of bloody feces. The lesions consist of a thickening of the intestinal mucosa. There may be sloughs of variable size in the involved mucosa and hemorrhages. Food particles have a tendency to adhere to the mucous membrane.

Prognosis

A guarded prognosis should be given. If the infection is relatively mild the percentage of deaths will be low but the economic loss due to loss of condition in the affected animals may be extensive.

Control

Sanitary principles are frequently ignored by the hog feeder and breeder. Although infectious necrotic enteritis may occur in swine that are maintained in clean pens and properly housed it is far more prevalent in swine that are kept in mud and filth. There is a tendency for the larger feeders to keep too many hogs in one lot, a fault that is conducive to the rapid spread of any infectious disease including infectious necrotic enteritis. The most successful swine husbandmen watch their herds carefully and at the first indication of any defect or disease isolate the affected animal. Quarantine is the first principle in controlling the spread of infection and to be most effective isolation should be absolute.

Rigid enforcement of quarantine regulations in the early stages of infectious necrotic enteritis materially diminishes the losses occasioned by this disease. Slop foods of good quality should be provided for affected animals. Tankage or other feed of high protein content should be withheld. The pigs should be kept in comfortable quarters and the droppings gathered daily, disinfected and properly disposed of. All dead animals should be burned, or should be buried sufficiently deep and limed to destroy infection.

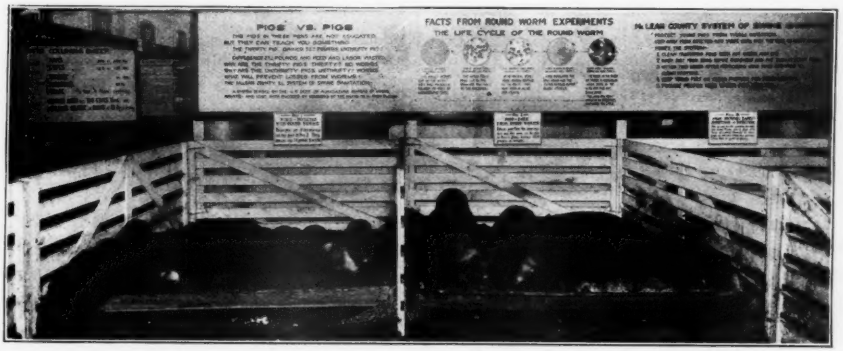
Prevention

The *S. suipestifer* and related organisms probably do not inhabit the intestine of normal swine; therefore, their presence signifies a carrier or a diseased animal. The organisms are eliminated in the fecal discharges and soils; feed and water are thus contaminated. Pens, lots, sheds and runs that have been occupied by diseased animals should be thoroughly cleaned and disinfected before other animals are placed therein. Since intestinal parasites predispose to infection with *S. suipestifer,* all swine should be periodically "wormed out".

Various biologic agents of reputed value as immunizing agents are available. *S. suipestifer* produces an endotoxin and it is probable that an opsonic immunity can be produced by a bacterin made from it. Reports of the successful use of such a bacterin are to be found in practically all veterinary journals.

Treatment

Affected swine should be provided with a limited ration of easily digestible alkalinized slop foods and placed in comfortable quarters. Intestinal antiseptics are indicated. The antiseptic selected should be such as can be administered in the slop.



From an exhibit of the U. S. Department of Agriculture. The worm-infested pigs, at left, averaged 54 pounds in weight; the worm-free pigs, at right, averaged 105 pounds at the same age. Worms and Salmonella organisms are often bed-fellows in the pig.

Ecto-Parasites and Mange Mites of Swine

The examination of the skin forms an integral part of the clinical examination of swine. The condition of the skin of an animal is a fair index to the condition of its health. The normal skin possesses a certain power of self-purification, that is, the healthy skin appears to be able to rid itself of many of the parasites that may happen to migrate to it. However, when an animal is suffering from some general disturbance of health, or is run down in condition, then certain of the ectoparasites seem to be able to multiply unrestrictedly. Parasitism in swine is, therefore, a problem of poor swine husbandry.

Unfortunately, the injurious effects, produced by these parasites, are not particularly spectacular, and therefore the economic importance of the ectoparasites of swine is often underestimated.

Myiasis

The screw worm, the larval stage of *Chrysomyia macellaria,* causes considerable damage and even fatalities in swine in some sections of the United States (Middle and Gulf States). The principal factor contributing to invasion is the soiling of the skin by secretions and defecations. The adult females deposit their ova in wounds and in other sores. The ova hatch in from one to ten hours, and immediately begin to burrow into the surrounding tissues. In addition to the damage from the mechanical laceration of the tissue, there exists the further harm from micro-organisms carried into the tissues by the larvae. The slightest and simplest injuries become transformed into serious ulcers with great loss of tissue, and with little tendency to heal. The larvae remain in the tissue from four to six days, then emerge and drop to the ground, where they undergo metamorphosis. Howarth has demonstrated that bone oil (by-product of the manufacture of animal charcoal) applied to wounds, is an effective safeguard against myiasis. The Bureau of Entomology of the U. S. Department recommends pine oil for the same purpose. A mixture of turpentine one part, phenol one part and olive oil eight parts is also effective in protecting wounds from screw worm flies.

The application of benzol to infested wounds, if evaporation is prevented results in destruction of larvae near the surface and thus lessens the number of screw worm flies in the next generation, and is thus a worth while prophylactic measure, although of limited therapeutic value.

Simuliidae

The black flies or gnats, members of the genus Simulium, are annoying pests, that have been known to attack swine. H. Osborn relates that hogs show the effects of the bites very little at first. Yet, large numbers die soon after the attack, while others die about six weeks after the disappearance of the gnats. The pigs usually perish from large ulcerating sores which result in septic infection. It should be kept in mind that certain species of Simulium are intermediary hosts of protozoan and filarial parasites; there is also the possibility that simuliid flies may play a rôle as mechanical carriers of bacterial germs. Livestock freshly imported into sections abounding in gnats are more apt to suffer and die from the bites than the native stock. Later in the season, the animals seem to develop a certain tolerance toward the noxious effects of these insects.

Lice

Hematopinus adventicius chinensis is a sucking louse. The fully grown female often attains a length of 3 to 4.5mm. The entire life cycle is passed on the host. Favorite locations for the deposition of eggs ("nits") are the hairs around the neck, shoulders, and flanks. The eggs hatch in about two weeks. The favorite location for

young lice, especially during cold weather, is the inside of the ear, where they congregate often in large clusters. The young lice reach maturity and mate when about ten days old. Two days later the females begin to lay eggs. In obtaining their food the lice puncture the skin of the host whenever a suitable place has been found, and begin to suck blood and lymph until satisfied. After each feeding they detach themselves and wander about through the hair. As each louse feeds at frequent intervals, and as a new puncture in the skin has to be made at each feeding, and as each puncture causes considerable irritation and itching, this annoyance is bound to interfere with the general health in cases of severe infestation, aggravating any illness that may be present. The frequent rubbing destroys the bristle and causes eczema and sores.

Chigoe—Chigger

In certain sections of the United States (Ohio, and likely farther South) the so-called Jigger-flea, *Tunga penetrans,* is occasionally found as a temporary parasite on pigs. The fertilized female pierces the skin and sucks blood until its abdomen begins to swell up, due to the growth of the fertilized eggs. As soon as the eggs are expelled, the female withers, falls away, or is expelled by ulceration. The season during which swine are attacked is short.

Sarcoptic Mange

Sarcoptes scabiei, var. *suis,* is a minute mite, roughly circular in outline, measuring about ½ by ⅓ mm; it is characterized by the presence of suckers. The life cycle of this parasite is imperfectly known. The female burrows into the skin and lays 20 to 40 eggs in the tunnel it forms. The larvae hatch in about three to seven days. The larvae, nymphs, young females, and males live on the surface of the skin beneath scales and crusts. After the mating of the male and the pubescent female, the latter becomes ovigerous. The whole life cycle is believed to require about two to three weeks. To obtain food, the parasite pierces the skin, sucking lymph; young epidermal cells are also occasionally ingested. The activities of the mites produce a marked irritation which causes intense itching and scratching, thus aggravating the condition. The parasites are found usually on the head in the region of the eyes and ears, or on the hind quarters. In later stages the mange spreads to the back and sides of the body. The inner surface of the thigh and the abdomen are rarely invaded. The symptoms of mange are redness of the skin, and the appearance of small nodules. In the course of time the skin becomes wrinkled and thickened, and covered with scales and scabs. The bristles fall out; itching grows intense. Underneath the crusts the skin is raw and cracked, and given to bleeding. Because of its contagiousness and its destructiveness to the skin, and because of the restlessness it produces in the animals infested, sarcoptic mange is a most serious disease, increasing in severity as it advances. Not only does it prevent proper growth and fattening of hogs, but it sometimes causes death in young pigs.

Demodectic Mange

Demodex phylloides is a leaf-shaped mite, about ¼ mm. in length. The mites penetrate into the hair follicles and sebaceous glands, where the entire life cycle is completed. Places of predilection are the

Demodectic Mange

region of the snout, and the lips, the inner side of the thigh, and the flexor surface of joints. In the early stages of the disease the affected skin may be red and scurfy; later the sebaceous glands become enlarged and appear as yellowish gray nodules, varying in size from a millet seed to a pea. Often pus formation sets in, leading to ulceration, and finally resulting in the falling out of the bristles. Itching is not pronounced.

Treatment of Parasitisms

The methods suitable for the treatment of lousiness and mange in swine are either hand application for individual animals, or mechanical application for mass treatment. The ideal parasiticide against either lice or mites is crude petroleum. It may be used undiluted or diluted with equal parts of cottonseed oil, or mixed with three parts of lard. The parasiticide should be applied either with a brush, or a mop, or a cloth. The remedy should be distributed over the entire surface of the skin in a thin, even

coating. When groups of pigs are to be treated it is expedient to bunch the animals together in a small enclosure and spray them with a mixture of crude oil or used motor oil, thinned with the necessary amount of kerosene, to make it spread easily. Allowing the animals to mix together for a short time facilitates the distribution of the oil over their bodies when crowding about. Spraying is somewhat less expensive than dipping, but it is also less effective, because the parasiticide does not reach all the surfaces of the skin, especially not the inner surfaces of the ear and of the thighs.

Another convenient method of mass treatment is by means of mechanical oilers. This method possesses the same disadvantages as the previous one. The simplest form of mechanical oiler consists of rubbing posts about which old ropes soaked in crude oil are wound. In moderate infestations this measure will suffice to hold the lice in check but not eradicate them.

The most efficient method for individual as well as for mass treatment is dipping the animals. For this purpose a vat may be used, or a special concrete lined tank may be built. A variation of the latter is the medicated hog wallow.

Oil dips are usually the most economical and dependable for eradicating lice and mites. A rational formula for an emulsion dip is as follows:

Hard soap	8 ounces
Kerosene oil	4 gallons
Water	2 gallons

M. et ft. emulsio.

The coal-tar-creosote dips, diluted in accordance with instructions by the manufacturer are suitable parasiticides. Two dippings 18 days apart are required for lice and three dippings at 14-day intervals are needed for mange.

When separated from the host animal, the lice live only two or three days; mange mites, likewise, are unable to survive for long when not in contact with the skin. Practically all cases of infestation occur from contact with lousy or mangy animals, and not from premises. Pens, corrals and hog houses which have contained lousy or mangy hogs are, therefore, not a source of danger to hogs free from lice or mange for more than a few days. Nevertheless, as a precautionary measure, such inclosures should be cleaned and disinfected before being used for other hogs.

As previously stated parasites of swine are a concomitant of poor swine husbandry practices and any treatment must contemplate better feeding, improved housing and a less insanitary environment.

The Swine Industry

Of the introduction of hogs into this country, the "Veterinary Military History of the United States," says:

"Swine were first introduced into the territory that is now the United States by De Soto, who brought a number of animals from Cuba to Florida in 1538. The swine brought to the northern colonies came from England and The Netherlands. They are described as being 'long-legged, large-boned, slab-sided, flop-eared, unprofitable and unsightly beasts better calculated for sub-soiling than for filling a pork barrel.' They were habitually permitted to run at large in the country and to some extent in the cities."

Swine raisers have made great advances in breeding and feeding since colonial times, but the control of disease has not kept step with the progress made by the geneticists and the nutritional experts. Swine disease control is probably less effective at present than it was 150 years ago; despite the fact that veterinary research in swine pathology has long since mapped out a sufficient sanitary program.

Notwithstanding the selection of brood sows for fecundity over a long period, the farmer still requires 19 sows to produce 100 pigs of weaning age. That is, in effect, one-third of his sows are "boarders"—live on his bounty, rendering him no return.

Of all pigs farrowed, one-third are born dead or die before weaning time and another third are so stunted by disease and mismanagement as to make no profit for the owner. Only one-third are sold at a profit. This, in the main, explains why most swine raisers constantly balance precariously between survival and bankruptcy, while at the same time, the consumer must pay a high price for pork.

Insanitation in live stock production has all along reached its culmination in the swine industry, and, as a result, the industry must write into its ledgers millions upon millions of dollars loss each year from a multitude of infectious diseases and parasititsms.

Apparently those in official charge of livestock sanitation in the states also have reached the acme of insanitation, if not the zenith of insanity in livestock disease control, in the methods they have employed or permitted to be used for the control of diseases of swine. It were unbelievable, were not the records indubitable, and it must ever remain unexplainable, that 22 (or more) of the states should have cultured hog cholera virus and distributed it indiscriminately to their swine raisers. No equally elaborate scheme for the distribution of infectious diseases among animals has ever been approached in the history of the livestock industry. The only parallel is to be found in the action of the benighted Hindu, who casts his dead of cholera and other infections, into the sacred waters of the Ganges and then drinks thereof to purify his soul and, incidentally, contaminates his body.

The swine industry has been saved from annihilation during the official distribution of hog cholera virus to farms in every township in some states, by the barrier interposed by the private veterinary service, which vaccinated so many hogs against cholera that the spread of the artificially produced outbreaks was blocked by immune herds.

The official, unrestricted distribution of hog cholera virus has been discontinued, but distribution, to a lesser degree, is still continued by a number of what is euphemistically termed "direct-selling serum companies." The states, in most instances, have full authority to prevent this and the United States Department of Agriculture can, at any time, stop this uncontrolled transportation of hog cholera virus across the state lines. That this type of insanitation (and insanity) is permitted to continue, is scarcely less excusable than the systematic spread of the disease earlier, under the guise of controlling it.

Our swine industry needs and deserves a better veterinary service than it is getting from the live stock remedy concerns, pill peddlers, bulletin doctors, county agents and other unqualified persons, who are supplying about three-fourths of its veterinary service. Our swine industry desperately needs an extension of that service of experts who, at the present time, are able to do little more than limit the harm done by the untrained. After 30 years' experience with anti-hog-cholera serum, cholera still costs the swine industry of the United States $500,000 per million hogs marketed. In Canada, where the control of hog cholera is placed wholly in the hands of veterinarians, the cost is $175 per million hogs, to cite a single example of the economy of relying upon experts in the matter of disease control.

Useful Prescriptions for Swine

Anti-Diarrheic

℞ Liq. Ferri sesquichlor 2.0
Mucilag. gummi arab..... 50.0
Tinct. opii simpl. 0.5
Aq. destill. 100.0
M.
Sig. A tablespoonful every 3 hours.
(For hemorrhagic gastroenteritis)

Anthelmintic

℞ Olei chenopodii .....ʒviij
Olei terebinthinae.....ʒj
Olei ricini.....ad Cong. j
Red coloring matter q.s.
M.
Sig. Give 1 ounce to each 50 lb. of animal weight.

For pigs with ascarid infestation. Approximate cost $5.00 @ gallon, or 4c per 50 lb. pig.
(E. W. Peck.)

Antidote for Phosphorus Poisoning in Swine

℞ Olei Terebinthinae ozonosatae.....
.....25.0 (to 50.0)
M.
Sig. Give at one dose.

Antispasmodic

℞ Chloralis hydratis 5.0
Lini sem. macerat. (5%).....100.0
M.
Sig. Give as an enema.

Aphrodisiac

℞ Cantharid. pulv. 1.0
Lauri fruct. pulv. 20.0
Piper. nigr. 4.0
M. ft. pulv. Div. in p. aeq. No. II.
Sig. One powder every hour.

Appetizer

℞ Olei phosphorati .....1 dram
Olei morrhuae q.s.....ad 10 ounces
M.
Sig. A teaspoonful daily.
(For a pig with malnutrition).
(Milks.)

Cardiac Stimulant

℞ Ol. camphorat .....30.0 (to 50.0)
Aetheris 10.0
Sig. For subcutaneous injection (2 to 5cc).

Emetic for Swine

℞ Tartar. stibiat 1.0
Ipec. rad. pulv.....3.0
M. et ft. electuar.
Sig. Give at once.

Emetic for Swine

℞ Veratrini 0.03
Spiritus 5.0
M. et ft. sol.
Sig. Inject subcutaneously.

Emetic for Swine

℞ Zinci sulphatis .....1.0 (15 grains)
Sacchari .....6.0 (1½ dram)
M.
Sig. Give at one dose.

Emetic for Swine

℞ Ipecac. pulv. .....2.0 (½ dram)
Veratri pulv. .....1.0 (15 grains)
M. et ft. chart. No. II.
Sig. Give one powder and repeat in one hour if necessary. — *Brumley.*

Expectorant

℞ Ammon. chlorid. 10.0
Glycyrrhizae pulv. 50.0
Mellis crud. q.s. ft. electuar.
Sig. A tablespoonful every three hours. For a pig with bronchitis.

Expectorant

℞ Ammon. chlorid. .....10.0 (2½ drams)
Sod. sulphat. .....60.0 (2 ounces)
Glycyrrhizae pulv. .....45.0 (1½ ounce)
M. et ft. chart. No. V.
Sig. Give one powder every 12 hours. To relieve bronchial catarrh. —*Brumley.*

Febrifuge

℞ Quininae sulphatis 1.5
Spirit. dilut. 35.0
Acid. hydrochl. dil. 5.0
M.
Sig. Give at once.

Laxative for Swine

℞ Magnes. sulphat. 100.0
Fruct. Anisi 50.0
Syrup. simpl. q.s. ft. electuar.
Sig. Give in one day. Laxative for a sow with mastitis.

Laxative

℞ Jalap. tuber.
Tartar. depurat. .....aa 2.0
M. et ft. pulv.
Sig. Give at once.

Laxative

℞ Hydrargyr. chlorid. mitis.....1.0
Amyli 0.5
M. et ft. pulv. d. t. d. No. IV.
Sig. One powder every two hours, until laxative action.

Mange Treatment

℞ Sulph. sublimat. 10.0
Ol. terebinth. 80.0
M. ft. liniment.
Sig. Apply externally.

Mange Treatment

℞ Liquor. Cresol. saponat. 50.0
Sapon. mollis 100.0
Spiritus diluti 100.0
Aquae 750.0
M.
Sig. Rub into the skin and repeat in 12 hours. Let 2 days elapse, and repeat again.

Mineral Supplement

℞ Calc. phosphoric. crud.
Juniper. fruct. .....aa 200.0
Sodii chlorid. 300.0
Ferri sulphat. crud. 50.0
M. et ft. pulv.
Sig. A spoonful in the feed mornings and evenings. (For pig with rickets or osteomalacia).

Narcotic

℞ Chloralis hydratis.....5 to 10.0
Sem. Lini decoct. 150.0
M.
Sig. Give as a clyster, to induce narcosis.

Plastic

℞ Ferri sulphat. 25.0
Sod. chlorid. 250.0
Carv. fruct. pulv. 50.0
M.
Sig. One teaspoonful to each pig as a blood plastic.

Protective

℞ Acid. carbol. liq. 2.0
Vitell. ovorum 2.0
Ol. Lini 60.0
M.
Sig. Apply to the skin.
(For swine-pox lesions).

Roundworm Treatment

℞ Santonini .....8 grains
Hydrarg. chlorid. mitis.....5 grains
M.
Sig. Mix well with the feed; give two doses daily for two days, then skip a few days and repeat. For 100 pounds of swine.

Sedative

℞ Urethani 10.0
Syrup. simpl. 20.0
Aquae .....ad 100.0
M.
Sig. 3 to 5 tablespoonfuls daily.

Sedative

℞ Urethani 30.0
Aq. Anisi .....ad 200.0
M.
Sig. A spoonful morning and evening.
(For a pig with convulsions).

Sedative

℞ Camphor .....3.0 (45 grains)
Sodii bromid .....1.0 (15 grains)
Sacchari .....1.0 (15 grains)
M. et ft. chart. No. III.
Sig. Give one powder and repeat in one hour, if necessary.
(For thumps—spasm of the diaphragm)
—*Brumley.*

Somnifacient

℞ Chloralis hydratis .....15.0 (½ ounce)
Glycerini .....30.00 (1 ounce)
M. e ft. sol.
Sig. Give as an enema, to induce narcosis.
—*Brumley.*

Stomachic

℞ Creosoti
Liq. ammon. caust. .....aa 5.0
Spirit. dilut. 150.0
M.
Sig. A tablespoonful every 10 or 15 minutes.

Stomachic

℞ Acid, hydrochlorid. dil. 2.0
Aquae destill 180.0
Rubi idaei syrup. 25.0
M.
Sig. A spoonful every 4 hours daily.

Stomachic

℞ Tartar depurat. 10.0
Sodii chlorid. 25.0
Fruct. Anisi 50.0
M.
Sig. A spoonful to each feed.

Stomachic

℞ Sal. Carolin, factit. crystallisat.
Glycyrrhizae pulv. .....aa 50.0
M.
Sig. A spoonful to each feed.

Plant Poisoning in Swine*

Swine are very generally believed to be quite tolerant to certain poisons harmful to other animals. The fact that hogs do not live on green plants to the same extent as cattle and horses, has led swine raisers in many instances to suspect other agencies as being the cause of death in cases of true plant poisoning in hogs.

That a very considerable number of plants common in the fields and pastures and in the hog lots of this country are poisonous to swine when eaten, has been amply substantiated by field cases and controlled experiments. It is probable that healthy hogs that are properly fed, seldom eat injurious plants; but, on the other hand, it is also certain that in the early spring when green forage is scarce, in cases of malnutrition, and when kept in dry lots and fed an unbalanced ration, hogs do occasionally, when opportunity offers, ingest plants that result in poisoning and death. The loss from plant poisoning in swine in the aggregate each year amounts to a very large sum.

Belladonna poisoning.—Atropa belladonna may be looked for in the United States as the cause of poisoning in hogs where the plant is cultivated. The symptoms are vomitus, excitation, convulsion, dilation of pupils, and blindness. In cases of sudden death, distention of the bladder is the only abnormality found. If the course of the poisoning is prolonged, gastro-enteritis will be found.

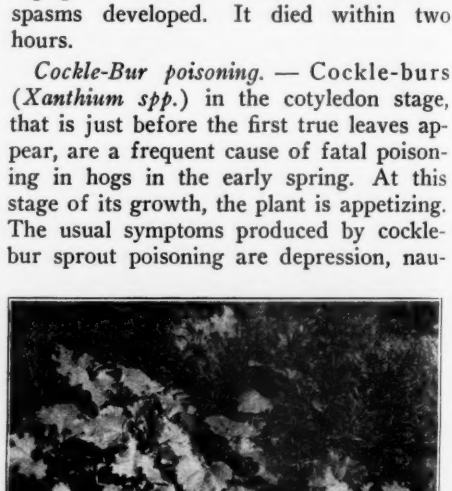
Black-eyed Susan (*Rudbeckia hirta*) *poisoning.*—When grazing on the roots and tops of this plant, hogs begin to display loss of consciousness followed by awakening and aimless walking for hours.

Buckeye poisoning.—Buckeye (*Aesculus glabra*) sprouts and nuts are poisonous to hogs. The poisonous principle apparently acts on the nervous system and causes paralysis, incoordination of movement, labored breathing, twitching of the muscles, dilation of the pupils, slobbering, and coma preceding death. The appetite seems to be unimpaired during the progress of the disease.

Buttercup poisoning.—Buttercup (*Ranunculus sceleratus*) frequently found in wet soil, contains the poisonous principle "anemonol," fatal to hogs.

China Tree poisoning.—The China tree (*Melia azedarach*), commonly cultivated in the South has compound leaves and purple flowers and a leathery, fleshy drupe. Hogs often have access to the plant, and undoubtedly frequent cases of poisoning occur. In an experimental feeding trial of green fruit pulp, a 90-pound pig exhibited a staggering gait 12 hours later; soon muscular spasms developed. It died within two hours.

Cockle-Bur poisoning. — Cockle-burs (*Xanthium spp.*) in the cotyledon stage, that is just before the first true leaves appear, are a frequent cause of fatal poisoning in hogs in the early spring. At this stage of its growth, the plant is appetizing. The usual symptoms produced by cocklebur sprout poisoning are depression, nausea, unsteady gait, spasms, labored breathing, rapid and weak pulse. The course of the disease is from 12 to 24 hours. If only a small quantity of the fleshy first pair of



Xanthium Canadense (Cocklebur)

*Condensed and revised from a discussion by the late Prof. L. H. Pammel.

leaves (cotyledons) is consumed, then the disease is less severe, and the affected animal may show evidence of colic pains, and slight diarrhea, but ultimately recover. In such cases there is rapid loss of flesh, and it takes several weeks for the animal to regain its former condition. If large quantities of the leaves are consumed in a relatively short time, a pig may die within a few hours. Cockle-bur sprout poisoning has frequently been confused with hog cholera and hemorrhagic septicemia. The use of fats, oils, whole milk, or cream is beneficial and may save poisoned animals, provided these antidotes are administered as soon as the hogs are observed eating these plants.

Cockle-bur is widely distributed in the United States from the Gulf of Mexico to Canada, and clear across the continent. The species, however, are not all the same. The common cockle-bur is a coarse, rough annual from one to three feet high. The stems are marked with brown spots; the leaves are rather large and heart-shaped with a long petiole. There are two kinds of flowers, staminate and pistillate, on the same plant in different heads. The fruit is covered with prickles and is two-beaked. The bur contains two so-called seeds. Sometimes some or many of the burs are pulled up and eaten with the young plants causing an acute gastritis.

Coffee Bean poisoning. — Sixteen pigs died with convulsions soon after being placed in a lot where coffee bean (*Cassia tora*) plants were the only vegetation. Autopsy showed an inflammatory condition of the stomach, and an enlarged, hardened, brittle liver. The stomach was distended with leaves of this plant.

Cowbane poisoning. — Poisoning from musquash root, poison parsnip, or spotted cowbane (*Cicuta maculata*) in hogs is of more frequent occurrence than is usually supposed, especially in some parts of the country where the ground is low. Our native cowbane is a tall, erect, branching herb with hollow stem, and large compound leaves, and white flowers in umbels. It has a pungent odor. Roots are fleshy and fascicled. Poisoning usually occurs during drought when hogs root after the fleshy tubers. The poisonous substance is cicutoxin. The symptoms generally begin with

Cicuta Maculata (Cowbane)

frothing at the mouth, followed by uneasiness and pain, and later by violent intermittent convulsions.

Crotalaria spectabilis poisoning.—Crotalaria spectabilis is a leguminous plant, widely used in Florida and other southern states as a cover crop and to improve the fertility of the soil. The green foliage of this plant is poisonous to hogs. The lesions found on autopsy are a severe anemia, accumulation of fluid in the abdominal and thoracic cavities, a firm, indurated liver, ecchymoses upon the endocardium, and gastritis, the latter often of an hemorrhagic character.

Dog Fennel poisoning.—Dog fennel (*Anthemis cotula*) is an acrid, branching, scented annual, one to two feet high. The

plant acts as an irritant to the gastro-intestinal tract in hogs.

Elder poisoning.—The roots of the elder (*Sambuca canadensis*) are poisonous to hogs.

Hemlock poisoning.—The poison hemlock (*Conium maculatum*) is a biennial herb, three to six feet high with spotted, branched stems, large decompound leaves with lanceolate pinnatifid leaflets. Flowers in umbels, white. Fruit small, somewhat flattened at the sides, smooth with prominent wavy ribs, without oil tubes; the face of the seed deeply and narrowly concave. All parts of the plant contain the poisonous substance coniine, but the seeds and leaves are the parts quite likely to be eaten. A. Hansen has reported cases of poisoning in hogs from this plant.

Henbane poisoning.—Black henbane (*Hyoscyamus niger*) is a clammy, pubescent biennial or annual which grows from one to three feet high; it has ovate, sinuate, toothed leaves. The flowers are on short stalks, dull yellow in color, with purple veins. This weed has been commonly naturalized in some sections of this country (as Utah). It is found from New England to the Rocky Mountains. It contains hyoscyamine and hyoscine. This is one of the most poisonous of our plants. It causes spinal paralysis, retards heart action, loss of muscular power, stupor, dilation of pupils. Death occurs from paralysis of the respiratory centers.

Jimson Weed poisoning.—Jimson weed, or as it is sometimes called, Jamestown weed is an extremly common naturalized plant in many places in northern United States. There are two species rather common, *Datura stramonium*, and *D. tatula*. These are rank-scented, tall, narcotic herbs with funnel-formed white flowers; both are annuals. The stramonium has a purplish stem; the tatula a greenish stem. Both of these species contain atropine, hyoscyamine, and hyoscine. Jimson weeds are commonly found in vacant lots, especially in hog lots. The symptoms of poisoning are vertigo, pain in the bowels, vomiting, nausea, extreme thirst, dry burning skin, nervous condition, dilated pupils, loss of sight, sometimes mania, convulsions, frothing of the mouth, and death. Hogs seldom eat the plant; probably the seed, which does not have the rank odor of the rest of the plant, is the part eaten, and causes the poisoning.

Mandrake poisoning.—The common May apple or mandrake (*Podophyllum peltatum*) is a perennial herb with running, yellowish rootstocks, a large, peltate leaf, white flower, and a yellow, fleshy fruit when ripe. The rootstock and the green leaves contain several poisons. Hogs that had partaken of the leaves and stems of the plant exhibited convulsions, staggering gait, and rapid respiration. Autopsy revealed petechiae in the kidneys.

Matrimony Vine poisoning.—Enteritis from the ingestion of the matrimony vine (*Lycium halimifolium*) has been reported by Dr. R. A. Craig of Purdue University.

Meadow Cone poisoning.—Meadow cone (*Rudbeckia laciniata*) is a smooth, branching, perennial plant with pinnately compound leaves, and with flowers on long peduncles. The flowers are yellow, the central part or disk greenish yellow. The plant is especially common in low grounds. It is on record of being poisonous to hogs. The symptoms are frothing at the mouth, eyes watering, seeming blindness, and inability to help themselves.

Meadow Death Camas poisoning.—Many authors state that swine are not poisoned by meadow death camas (*Zygadenus elegans*). If this is true, it may be inferred that these animals do not suffer from the plant, because its emetic action disposes of the material so completely that no further symptoms appear. While it seems probable that the vomiting prevents serious poisoning, it may be possible that if the animals had eaten large quantities, enough might have been absorbed before vomiting to produce other symptoms and possibly death.

Night Shade poisoning.—Black night shade (*Solanum nigrum*) possesses white flowers and black berries. The leaves are ovate and stalked with a rather strong odor. The plant is commonly naturalized in the United States. The western night-

shade, known as the three-flowered nightshade is similar to the above, but is much more toxic in its action. The flowers are whitish, the berries are black, and occur three in a cluster. According to Kinsley, poisoning is not frequent. Animals that die within a few hours following ingestion, show no lesions at all, except retention of the urine, and a greatly distended bladder. The urine is mydriatic. The blood may be somewhat darker than normal. The first symptom noted is nausea, followed by vomiting, dryness of the mouth, more or less excitement and convulsions, increased pulse and respiration, dilated pupils and partial blindness. The duration of this period is short, and is followed by paralysis. Treatment consists in administering antidotes of atropine, emetics, and purgatives; in stimulating the heart, and in restoring respiration by artificial means. In some cases sedatives may be given.

Green Potato poisoning.—Most of the cases in hogs reported in this country were from places in which garbage was used. There are many cases of this kind on record. Garbage frequently contains green (sunburnt) skins of potatoes or potato sprouts. Ahlers also reports potato top poisoning in Iowa. In one instance an entire herd of hogs developed paralytic symptoms after feeding for some time upon a ration consisting almost entirely of raw potatoes that were more or less withered and sprouting. Cases of poisoning from semi-rotten potatoes have been reported in both horses and hogs (from tubers spilled at a freight car where they were being loaded). It seems probable the damage was done by sunburnt rather than decaying potatoes.

Poke Root poisoning.—Poke root (*Phytolacca decandra*) is found from New York through Ohio, Indiana, Southern Iowa, Missouri and Kansas and westward to Texas, and east to Florida. The berries are crimson; the flowers are greenish white, with large petioled leaves. Stems and leaves are more or less succulent, coming from a long, stout, tuberous root, blackish in color. All parts of this plant, particularly the roots and green berries are poisonous to hogs.

Red Root poisoning.—Red root (*Iachnanthes tinctoria*) of the blood wort family is a stout, tall herb with numerous yellow flowers, six-parted perianth, and few seeds. It is found in salty swamps near the coast in southeastern Massachusetts, Rhode Island, and New Jersey to Florida. Throughout the South, white hogs are supposed to be particularly subject to the poison contained in this plant. While the claim of immunity of black pigs from the effects of red root seems to be a common belief, investigation is required to confirm it.

Rhubarb poisoning.—The leaves of the garden rhubarb (*Rheum rhapontiacum*) contain dangerous quantities of oxalates. Nine hogs which had eaten a wheelbarrow full of rhubarb leaves died in from three to four hours, exhibiting convulsions. The earlier symptoms were foaming at the mouth, and a staggering gait.

Water Parsnip poisoning.—The water parsnip (*Sium cicutaefolium*) resembles somewhat water hemlock. The symptoms of poisoning in hogs are frothing at the mouth, trembling, rapid respiration, slow heart action, and bloating preceding death.

Senecio Integerrimus (Groundsel)

Coöperation Between Veterinarians and the S. P. C. A.

By WESLEY A. YOUNG, Chicago, Illinois,
General Manager of The Anti-Cruelty Society

THE dictionary defines the word "co-operation" as meaning, to operate together for a common objective, joint action or profit sharing. "Humane" is defined as having or showing kindness or tenderness, and "veterinarian" is defined as one skilled in treating diseases or injuries of animals.

I can never think of coöperation without remembering a little advertising card I once saw and I presume most of you have seen it. Briefly, this card showed two calves tied together with a length of rope. Two pails of milk were placed a little farther apart than the length of the rope. So long as the calves pulled in opposite directions, neither got any milk. Finally they coöperated—emptied one pail and then went to the other to continue their profit sharing by joint action.

It seems almost unnecessary to say anything to confirm the absolute necessity of coöperation between veterinarians and humane societies.

For purposes of interest and information, I would like to delve into the history of veterinary medicine for just a moment, and quote from a two-volume work on veterinary history published recently by Drs. L. A. Merillat and D. M. Campbell, both of Chicago.

"The birth of the veterinary art preceded that of human medicine. In biological existence, food is the primitive requirement. Veterinary medicine sustains life, human medicine preserves it.

"When man came upon the earth, his survival depended upon proving his superiority over the hordes of animals which had preceded him by several millions of years. That he captured, tamed, bred, and utilized the more tractable species, in order that the others might be subdued, is self-evident. That the undertaking was slow, dangerous, uncertain would not be contradicted by connoisseurs of wild animal life. The training process required cunning, kindness, skill, knowledge and invention with which there developed contemporaneously the arts of zoötechnics and veterinary medicine.

"In his adventurous struggle, animals became an indispensable possession of man; and providing shelter and food, maintaining their health, and improving their physical makeup has been one of his routine obligations. From the legendary past to the world of today, the importance of animals in the pursuit of comfort and happiness never changed with the rise and fall of civilization or nations. It is, therefore, academic, if not utilitarian, to set the birth of veterinary medicine at 4,000,000 years ago, or, if Genesis is accepted as the history of Creation, its cradle was the Garden of Eden.

"The first advances in woodcraft, house construction, warfare, agriculture and the chase were made with the aid of the large herbivores (horses, asses, cattle). The porcine, ovine, caprine and avian species were domesticated during more definite periods of human existence. It is evident that man did not domesticate animals until he reached a certain state of domesticity himself (Hough). The process was a gradual one, developed among the pre-historic people of Asia and Europe. The taming and utilization of animals is an art of Oriental origin. The North American Indian did not domesticate animals, and remained a savage, as did the natives of Australia and Africa, until the influence of a foreign people reached them. They hunted but did not train the fauna among them, useful as buffalo, elk, wolves, turkeys, ducks, geese, etc., would have been. During the same centuries that saw the forming of Assyria, Egypt, Babylonia, Greece and Rome, in the development of all of which domestic animals played a significant rôle, the arts of civilization made no headway on the continents where animals were not domesticated.

"All are in agreement that the domestication of animals was accomplished, not by force but chiefly by kindness: feeding the hungry, the distressed. The dog, generally conceded to have been the first animal to yield to the ministrations of man, came to him unsolicited as the scavenger of the settlements, a camp follower that acquired the habit of guarding the camps by chasing intruders from the garbage dump. From this stage it was but a step to sharing man's shelter and fire, assisting him in the chase and the close alliance of dog and man that has obtained throughout all of known history.

"In the transition from the precarious existence and poverty of the savage to the security and wealth of the civilized, from hunter to farmer to craftsman, the estate of man, of tribes, and of nations grew in proportion to the intelligence displayed in the husbandry of animals. The caveman, the pastoral nomads and the nations succeeding them prospered or vanished with their flocks. Animals swept away by plagues left their appalled possessors prey to predatory neighbors. Wealth

* Presented at the 60th Annual Convention of the American Humane Association, Houston, Tex., October, 1936.

and political power grew only where wisdom was shown in the care of their animal budgets. The successful warriors were the good horsemen who left bountiful herds behind to sustain them when they went forward to battle. A prolific animal husbandry has always spelled prosperity and power, a scrawny one, poverty and decay. In all great wars of history, the fate of the nations implicated has oscillated with the animals available to the soldiers and the civil population. The World War was no exception.

"The early husbands of domesticated animals were not long in discovering the pitfalls of their undertaking. Diseases unknown in wild life struck them with despair and gave birth to the magic, the prayers, the imprecations, the incantations and other rites practised as cures preceding the primitive studies of animal medicine. Of these we need not speak except to say that they lived too long.

"Diseases of animals are described in the earliest medical writings in a fashion indicating that knowledge of them was handed down from a distant past; doctors for animals were listed among the functionaries of most ancient governments; twenty centuries before the Christian era, laws regulating the practise of veterinary medicine were in force; carvings unearthed in archaeological research portray the care of animals in periods antedating known history.

"The Revolutionary War, which separates all American history from the colonial period, led to the development of a tremendous livestock industry.

"The Civil War gave birth to the veterinary profession, *ex necessitate rei* [in other words, "save the animal or perish"]. Notwithstanding that the annual production of domesticated animals exceeded 100,000,000, the food and clothing supply of the American People had become precarious in the absence of a system of disease control among them. A veterinary-medical organization was established to plan systematic disease control among animals and schools were founded to furnish the necessary personnel."

In the work of disease prevention among food producing animals nearly one-fourth of the graduate veterinarians in the United States are employed. Disease prevention I need not mention is the highest type of anti-cruelty or humane measures and the most effective. Do you realize that one-fourth of the veterinary profession is working every day that your food may be more free from disease than has been the case in the past? Such things as the reduction in the amount of tuberculosis among our dairy and beef cattle, as well as herds of swine and flocks of poultry, have been brought about by this army of veterinarians, working for the good of the entire population of our country. The work of this group of men is not very well understood and I believe is little appreciated by the average person, largely due to the fact that the public neither knows nor realizes the vast amount of effort put forth by these veterinary inspectors to safeguard public health and assure a regular and adequate food supply for the nation.

The public is much better acquainted with the remaining group of veterinarians, whom we know as the practitioners of veterinary medicine. They are the men who take care of your sick and injured pets.

You recall, in the quotation, various references to the domestication of animals by *kindness* and *protection of the health.* These are two fundamental laws which have never changed down through the ages. Humanitarian activities and veterinary activities have been wrapped up in the same package ever since the days of creation. The real reason we have veterinary medicine is to relieve the suffering of animals. The first scientific veterinary work published in this country, (Clarke's Preventive Veterinary Medicine, 1793), pleads on nearly every page for a kinder treatment of animals and inveighs unendingly against the cruel customs of the time. The first veterinary journal published in this country (*American Veterinary Journal* 1852-58), more than anything else counseled a more humane attitude toward animals and endeavored in every issue to induce the owners of animals to discontinue cruel practices toward animals common in that day and particularly were they urged not to permit quacks of which there were many at that time, to practise the hideous cruelties upon the sick or injured animal for which quacks have ever been notorious. Likewise, the real reason we have humane societies is to relieve the suffering of animals. Their purposes are basically the same. A veterinarian practises his profession of relieving animals and is paid fees for it by his clients. You and I, as humanitarians, relieve animals and are paid a salary from the funds provided by philanthropic people. We are all working in the

same basic work, but supported in two different ways.

The practitioner of veterinary medicine, should he attempt to become a charity, will find himself operating at a financial loss and eventually must discontinue his work however good it may be. Consequently, he must be commercial in his activities. The humane society, being a philanthropic charity, is the logical place for relief for animals owned by people who are financially unable to pay for the practitioner's services. This is as should be. No individual can be expected to work without compensation. No owner of an animal should expect an individual to treat it gratis. Every animal has a right to the relief of its sufferings. Consequently, those persons financially able to do so should support the animal protective societies in order that they may provide veterinary medical relief to the animal companions of those unfortunate individuals unable to pay for such service.

I hope that animal protective societies throughout our country will establish clinics for the relief of suffering of all sorts of animals, birds, reptiles or what have you. But I truly hope that they do not go into the commercial practise of veterinary medicine. To do so destroys that joint action of which I spoke and certainly is not operating together for a common objective. Remember so long as the calves pulled in opposite directions neither of them accomplished anything worthwhile. Some of our existing enterprises are scarely justifiable. We, as humanitarians, should make a very definite effort to avoid increasing such questionable enterprises.

The humane societies throughout the country advertise the veterinary profession. This is due to the societies which are constantly telling the people: "be kind to your animals," "when your animal is sick or injured call a veterinarian or at least have veterinary services provided for the animal." All of these things are definitely in keeping with the definition of coöperation, which is joint action or profit sharing.

I find that as I contact the practising veterinarians throughout the country they are nearly 100% in accord with the fundamentals of animal protection. Many of them are members of our societies. They question a society's activities only when it forgets all thought of coöperation and becomes a commercial competitor.

The veterinarian can be, and in most cases is, a very great asset to an animal protective society. Here are some instances:

Clinics—Thousands of animals are relieved in these clinics that would otherwise go untreated, or submitted to the ministrations of quacks.

Diet—We all must depend upon the professional man for a proper diet, housing and general care of animals. True, anyone knows considerable about these things, but when the time comes that an argument or difference of opinion arises, or the unusual case comes up, the question is decided by the veterinarian.

Court Cases—It has been my experience that judges and juries place more credence in professional testimony than in all the other testimony presented. I would hesitate to go into court on many cases of cruelty without professional support.

When to destroy a dog or animal—especially a stray—The decision to kill or not to kill an animal should depend upon the physical condition, habits, etc., of that particular animal. These points should, if possible, be decided by professional help and not depend upon the whim or fancy of some person so often well meaning, but lacking in proper understanding of animal problems.

The veterinarian makes his principle contact with people at the time when they are thinking about their animal's welfare and comfort, due to the fact that the animal is sick, injured, or that the owner is in need of some specific advice. That is the ideal time; it is the psychological time to plant the seeds of "reverence of life." In other words, to plant our humanitarianism in the hearts of the public.

The longer I labor in animal protective activities, the more I am convinced that veterinary medicine and animal protection are inseparable. Every society for the protection of animals should have one or more competent veterinarians on its staff.

Treatment of Septic Conditions of the Phalanges

The general practitioner is frequently called upon to treat various diseases of the foot. One such disease is a chronic septic condition with necrosis involving the second and third phalanx bones and surrounding tissues. Usually such cases are brought to the veterinarian after the owners have tried various and sundry treatments—lime, iodine, alum, and other agents.

These cases usually extend over a period of months. They are caused by the animal's bruising its foot, or by some foreign object piercing the sole or frog. In my locality the farmers are troubled with sassafras bushes, which to be controlled must be cut with mowing machines two or three times a year, and I have under treatment each year many horses and mules which have stepped on the freshly-cut stubs. The stub pierces the sole or frog, and when it is removed, the thin bark usually remains within the foot, giving rise to an infection.

In treatment it is wise to take complete charge of the case for at least 30 days. It is my practice to keep the animal in my own stable for this length of time.

The foot is examined and the extent of infection determined as nearly as possible. In chronic cases the surfaces of the phalanx bones are usually involved. To alleviate pain and permit the operation to be performed in standing position, the digital nerves are blocked by local anesthesia. A large opening is made over the region of the old opening, and all necrotic tissue is removed. When the anterior surface of the foot is involved, the infection usually extends from the sole to the top of the hoof wall, and most of the pus discharge is at the top. When the posterior surface and the frog are involved, the frog is removed to obtain perfect drainage. After all diseased tissue is removed the surfaces of the phalanges are scraped.

The next step is to fill in with sound tissue the space left by the removal of the diseased. For several days I run through the opening one gallon of Carroll-Dakin solution, setting the tube so that it takes about one hour to use the gallon. Each day in examination some small areas of diseased tissue are found and removed. After five or six days of this treatment, I begin the use of allantoin, which in my experience is one of the most beneficial drugs for this purpose.

This is a necrosis of a phalanx. The frog has been removed. Note the steel plate for protection.

Allantoin is an excretory product of maggots and is responsible for the beneficial results obtained in maggot therapy. Since it can be prepared synthetically, the synthetic product can be used in place of living maggots. It is available in capsule form, for making one's own sterile solution, or it can be purchased in solution ready to use.

Each day the cavity in the foot is packed lightly in sterile gauze moistened with allantoin solution. Another moist pack is applied over the top opening, and a light bandage applied. This dressing should be changed daily. After a few treatments small, shining, pinkish, granulating areas begin to grow in the wound. This tissue bleeds easily and has the appearance of healthy tissue. The packing gauze is reduced in amount each day as the wound closes and, usually in three to four weeks, the wound is completely healed. The animal is then turned back to the owner with instructions for care. I usually dispense a urea powder to be used daily.

The cost of this treatment is low. The allantoin capsules (6.17gr) can be purchased at $2.70 for 25. This quantity makes 2500cc of 4% solution; 25 to 35cc used daily is sufficient.

VILO T. ROSE.

Elkton, Ky.

Drugs Used in Veterinary Medicine

XI. INSECTICIDES. Part 2*

By VICTOR LEWITUS, New York City

PYRETHRUM

Historical Sketch

It has long been recognized that certain members of the daisy family (Compositæ) possess insecticidal properties. Pyrethrum flowers have long been used in their original habitat, Asia Minor, for such purposes; but it was not until recently (about 100 years ago) that large scale cultivation was introduced into Dalmatia along the Adriatic coast. The first recorded importation into America took place in 1860. Because of the great popularity that this most useful product attained, and also curtailment of the supply as a result of the Great War, Japan became the most reliable source of supply. Today, about 80% of the world's supply is obtained from Japan. There is some promise of production in the Kenya District of East Africa and even in California, but as yet this source cannot be depended upon. For general agricultural and veterinary purposes, the United States consumes around 15 million pounds annually.

A few interesting side-lights on the history of this plant will be cited. According to MacOwan, the nature of insect powder, as the preparation was known, was discovered by the Russian military authorities in the Caucasus through some prisoners. The cantonments were swarming with fleas and it was noticed that only a powder prepared from certain species of plants (later found to be pyrethrum) were really effective. This secret had been carefully guarded by the natives. Dalmation insect flowers were not popular before 1854, probably also because of the unwillingness with which the natives divulged information regarding a product which they wished to monopolize. Furthermore, the growers were careful to roast the seeds they sold or treat them otherwise to prevent germination. It was Jüttner who reported the original discovery of the effect on insects in 1840. A native Ragusan woman (in Dalmatia) picked some flowers which she intended to use for decorative purposes. Once the plants became withered, she cast them aside into one corner of the room, and to her astonishment, discovered later a large number of insects near them stone dead! She concluded that the death of the insects must have been due to some virtue possessed by the flowers. She was not wrong, and to her dying day engaged in the production of the plants, thus "cashing in" on her casual observation.

Sources, Principles, Chemical and Biological Assay

There are numerous sources of pyrethrum or "insect flowers", those recognized by the United States Department of Agriculture being three: *Chrysanthemum* (section Pyrethrum) *cinerariifolium, Chrysanthemum* (section Pyrethrum) *roseum,* and *Chrysanthemum* (section Pyrethrum) *carneum* (or Marshallii). The first-mentioned of these is recognized as the best commercial variety. These flower heads are collected when in full bloom, though until recently it was not determined when their toxic principle was present in greatest amount. Since the heads are picked along with the stems, the latter—formerly purposely added as a sophistication—must be removed. The parts are allowed to dry and then the drug is ready for shipment.

The active principle of pyrethrum consists of two esters located chiefly in the ovaries or achenes (small nut-like fruits) contained in the late stages of flowering. The isolation of the active principle baffled scientists for many years, but finally two Swiss chemists, Staudinger and Ruzicka, isolated two related components which they named Pyrethrin I and Pyrethrin II. The flowers under examination were found to yield approximately 0.2% to 0.3% total pyrethrins, of which No. I was present to the extent of 40% and No. II, 60%. Later, Staudinger and Harder found as much as

* Note: The previous article (X) dealt with the well-known insecticides, sulphur and nicotine. The present one concludes this topic and includes the discussion of the drugs pyrethrum and derris (rotenone).

0.6% total; and since then workers have found ranges of 0.6% to 1.2% pyrethrins present. East African insect flowers are known to contain as much as 1.25% to 1.44% of these valuable esters. Tests on certain aphids (*Aphis rumicis*) have shown that in alcoholic solutions Pyrethrin I is about 10 times more toxic than Pyrethrin II.

These esters are organic compounds derived from the monovalent and divalent chrysanthemum acids. That the properties of the esters differ considerably is indicated by the fact that No. I distills in vacuuo at 150°, whereas No. II decomposes under identical treatment. The difference in relative activity has already been pointed out. Investigators suggest the possibility that the drug may owe its full activity to some as yet unidentified constituent.

Various chemical methods of assay have been proposed, chief among which in this country have been the following, in chronological sequence: Staudinger and Harder (1927) acid method; Tattersfield, Hobson, Gimingham (1929) improved acid method; Martin and Tattersfield ((1931) ferricyanide reduction test; Gnadinger and Corl (1928) copper reduction test. For some unexplained reason, none of the above-mentioned methods of chemical analysis show distinct correlation of killing power on insects. For this reason, an index of toxicity must be made from a biological assay. This tests out directly the effect on the insects themselves. Many methods have been described; the most practical and acceptable being the Peet-Grady method. Briefly described, the assay consists of subjecting the common fly (*Musca domestica*) to the effects of the drug. One hundred flies, bred in the laboratory, and five days of age, are placed in a chamber, six feet in each dimension, into which is introduced 12cc of the insecticide to be tested. The minimum standards require that 95% of the flies be "down" in 10 minutes, and 60% dead after 24 hours. Nine batches of flies are employed and the results are then averaged. It has been suggested by some that the death minimum should be raised to 70% or more, since a good grade of pyrethrum tests out 70-75% effective. An improvement of the above method has been devised by Campbell and Sullivan, in which a round table method, (using glass jars 20 inches high by 10 inches wide) has been found much more rapid in determinations. Nelson *et al* have recently found comparable results with their "direct application" method. A measured amount of the unknown insecticide is applied to the thorax of an individual fly which has been chilled in a refrigerator. The drug is applied by means of a finely-drawn-out glass tube. From 50 to 100 insects are employed.

Action

It was formerly believed that the insect inhaled or absorbed the dust, spray or fumes from this drug through the spiracles of the tracheæ located along its body covering or integument. This notion came about because we know that insects, unlike higher living organisms, breathe throughout their bodies. It was Wilcoxson and Hartzell in 1933 who demonstrated that pyrethrum kills mainly because its contact with the integument, independent of introduction through the spiracles, results in a paralysis of the nerve ganglia located therein. This has been proved time after time by studies on cross-sections of affected insects which indicate marked dissolution at these vital points.

It is known that whole flowers have relatively good keeping qualities, but once powdered they may deteriorate to a great degree. This deterioration averages about 5% progressive loss of toxicity each nine months. Freshly-milled flowers should be used in making up stock solutions. The pyrethrins in a pure state are known to decompose rapidly. The decomposition is due to one or more of three recognized causes: hydrolysis (due to water in the flowers or atmosphere), oxidation (from the air), and polymerization (the latter changing the pyrethrins into inactive compounds). Because of this degeneration, certain anti-oxidants are added to commercial preparations. In making up solutions of pyrethrum concentrates, inert waxes and resins are removed by refrigeration methods. The extractive is then dissolved in

alcohol, acetone, pine oil or some mineral oil (hydrocarbons).

Livestock Sprays

An effective stock spray should meet the following demands:

1. It must show immediate toxicity to insects present.

2. It must serve as a repellent to prevent further infestation.

3. It must be safe to the animal being sprayed.

A few formulæ* are as follows:

I.

Pyrethrum extract	1 gallon
Derris (liquid extract) (5 grams rotenone per 100cc)	½ gallon
Pine oil	2½ gallons
Liquid petrolatum	2 gallons
Base petroleum oil	54 gallons

II.

Pyrethrum extract	1 gallon
Pine oil	2½ gallons
Base petroleum oil	56½ gallons

The pyrethrum affects weaker insects such as flies and mosquitos promptly; whereas the derris affects hardier organisms even though it is much slower in action.

Some recent work has shown both pyrethrins to be effective in hog ascarides and dog tapeworm (according to Chevalier); and Puchet has shown that even a single dose influences Ascaris, Oxyuris, Trichocephalus, Ancylostoma and Tænia. According to Ricaud *et al*, pyrethrin in oil is effective in certain bovine bronchitis cases due to parasites. Fourneau *et al* found that it fails to influence the course of bird malaria.

It should here be pointed out that "Insect Flowers" must not be confused with pellitory root or *Anacyclus pyrethrum*, which was formerly used in human medicine as a sialogogue. This has no place as an insecticide, even though it is a closely-related plant.

Toxicology

Reports on the toxicity of pyrethrum flowers are quite variable. On warm-blooded animals it appears to be relatively harmless though on amphibians and fishes it appears to be very active, as with insects. Zeigler found the extracts toxic to warm-blooded animals when injected intravenously. Riley reports a case in which the powdered flowers were rubbed into the skin of a dog, after which the animal suffered effects in its locomotive organs as with insects. Carruthers reports a species of pyrethrum as affecting the digestive tract of stock by injury to the lining of the stomach. Coquillet reports that horses eating the stems of the plant were not injured. On small animals in enfeebled condition the drug is contraindicated since the pyrethrins are known to produce gastroenteritis even when small amounts are ingested by licking. Furthermore, a dermatitis may set in because of the resinous constituents in the crude powdered forms. In the case of infested kittens or puppies, it has been suggested that the powder be sprinkled onto the coat for about 15 minutes or less, and then brushed off after it has had its effects. Horning recently reported (VETERINARY MEDICINE, September, 1934, p. 387) cases of gastroenteritis in dogs and cats which were traced to the use of this insecticide as a dusting powder. Observation has shown this practitioner that the animals need not necessarily lick themselves; for toxemia developed just from shaking the powder on them. Gastroenteritis developed when the animal did lick itself. Dermatitis occasionally is traceable to the powdered flowers and these cases may be treated by prompt removal of the powder by bathing. Some cases have been found in the presence of other insecticides, such as "tuba-root", which lead quite often to gastric syndromes. Horning mentions that in all cases of gastroenteritis, he ascertains the nature of insecticides used on affected animals.

DERRIS ROOT (Rotenone)

Historical Sketch

One of the most effective of modern insecticidal agents is derris root. Only recently introduced into the white man's armamentarium, it had been used some centuries back by the natives of the Indian Archipelago and the Antipodes as a fish and arrow poison. To poison fish the natives poured water into their canoes and

*Courtesy S. B. Penick Company.

rinsed the masses of freshly bruised derris root in it. The milky solution was thus taken downstream and the boats overturned. In about 20 minutes, large schools of fish were seen to rush wildly in all directions. Using spears and clubs, the agile men made short work of them and the women scooped up their catch in large nets made of grass. What fish were not consumed on the spot, were dried for some future festive occasion. We always are prone to question the safety of such a repast, since the toxin acts so quickly; but if one considers the great dilution which will cause poisoning in these cold-blooded animals one is not so readily misled. Recent experiments have shown that as little as 1:13,000,000 of rotenone (from the derris) will kill goldfish. In fact, certain foreign laboratories use this animal for assaying rotenone.

The earliest reference to derris root appears to be in 1747 in a Dutch paper, which described it as a fish poison. Later (1848) its popularity was enhanced by its use in controlling larvæ found on Nutmeg trees. In 1902, Nagai isolated the active principle, rotenone, to which he assigned an erroneous chemical formula. This was later corrected by the French chemist Butenandt. The British improved research until today in that country, derris is used to control the warble fly, which had formerly punctured the hides of cattle and lowered their value when converted into leather. Over 100,000 cattle on nearly 500 farms were thus freed of this pest within the last decade. The Chinese learned to use the derris in a decoction to protect their truck crops, and American farmers soon came to an adoption of similar methods. In place of the customary dangerous arsenicals, more and more this very effective and relatively safe insecticide has made a place for itself. Likewise in veterinary circles, derris root and its extractives are more popular than many of the older "standbys".

Sources

In the Far East, the "tuba" plants are merely looked upon as convenient fish poisons, and this term has in some way crept into commerce. Derris root, one of these, comes from the plant known as *Derris elliptica*. This contains the highest yield of rotenone, its active principle; but there is another source in the "Cube Root" or *Lonchocarpus nicou*, which grows in Peru. Although this also is high in rotenone content, the present commercial preference lies with the East Indian derris plant. It is interesting to note that a plant native to America (*Cracca Virginiana* or Devil's shoe strings) contains rotenone, but its content is too low for present extraction on a commercial scale.

Rotenone

The most potent insecticidal principle in derris root is rotenone, but some other valuable components resembling nicotine in activity such as deguelin and tephrosin are present. Other items, such as toxicarol and dehydrorotenone are considered practically inert. Tests are still in progress on the non-crystallizable fractions of the drug.

Rotenone ($C_{23}H_{22}O_6$) is a colorless, odorless, crystalline substance; soluble in the following order: chloroform (very), ethylene dichloride, trichlorethylene, chlorobenzine, ethylene and chlorhydrin, benzine, acetone and toluene. In the lower aliphatic alcohols it is soluble in the ratio of 0.2 gram per 100cc; in kerosene, less than 1 to 2000; and in water about 1 to 1,000,000. Rotenone deteriorates in the presence of light, and probably also in air.

Assay methods for rotenone have been worked out in Washington on the basis of its insolubility in carbon tetrachloride and its ease of crystallization from a concentrated solution. More recently, methods of extraction with chloroform have been developed.

Application or Use

Derris root, and especially its extractive principle rotenone, appears to be compatible with pyrethrum and is considered to enhance its activity. While the "downing" effect is only slightly influenced, with rotenone the "kill" on flies tested by the Peet-Grady method is increased about 20%. Derris is a much slower but more powerful acting insecticide than pyrethrum. Since derris is not soluble in petroleum distillates, it must be added by means of previous

solution in hydrocarbons such as ethylene dichloride (1:2000). (For formula see under Pyrethrum—formula No. 1.)

For fleas and lice, a mixture of pyrethrum and derris powders (or rotenone) cannot be improved upon with present knowledge. Whereas pyrethrum is prompt in action, derris is slower but more certain to produce death to the insect pests without endangering the life of the host. Diluted with talcum or other diluent powder, powdered derris root is employed in dusting powder formulæ to give a 1% rotenone content. From 10 to 20% of powdered pyrethrum is added for additional insecticidal action. Such a combination is considered harmless to cattle, dogs, cats and poultry.

Crane of the New York State Veterinary College has found that 1/20 gram of rotenone per kilo of weight in dogs (followed by castor oil) effectively removes hookworms and roundworms. He also finds that it has great promise in treating cases of follicular mange. Out of 13 cases, 11 responded and progressed to complete recovery in one to five weeks. The other two cases, though cured, were under treatment with rotenone as with ordinary methods. He applied the following solution daily until cure was apparent:

Rotenone	1 gram
Alcohol	50cc
Acetone	10cc
Water	40cc

The rotenone should be dissolved in acetone before adding the rest of the liquids.

Toxicology

As with pyrethrum, it appears that rotenone or derris root is selective toward cold-blooded animals. It is highly toxic to fishes, crustaceans, reptiles and insects, but is practically harmless to man, fowl, cattle and other domestic animals. Buckingham found that in doses up to 1 grain per pound body weight in dogs when given by mouth, pure rotenone produces no visible effects. Haag found that in testing the material on guinea pigs, rabbits, dogs, cats, pigeons, frogs, and white rats, there seems to be little danger of acute intoxication in healthy animals when rotenone is ingested with food. He himself swallowed 150 milligrams (2.3 grains) without experiencing harmful effects. In aqueous solution the drug is harmless to mammals, but when dissolved in oils it may cause a temporary reduction of blood pressure. If injected directly into the bloodstream it produces irritation and swelling. Inhalation is known to cause temporary numbness of the respiratory tract. It should not be applied to cuts or allowed to be inhaled by animals for these reasons.

In large doses internally, rotenone affects the nervous system, causing excitement, increased respirations and heartbeat, and convulsions. Later there may be depression and incoördination, followed by paralysis. In the dog, salivation and vomiting may be a prominent symptom; and urination may occur involuntarily. Treatment in these cases can be only symptomatic since there is so much variation possible. Sedatives at first, and stimulants during depression are indicated. Evacuation of a slowly soluble drug such as this is also in order.

It is the writer's opinion that as time goes on the popularity of both pyrethrum and derris (or rotenone) for the treatment of cases of fleas or lice or certain types of mange will be progressively enhanced.

NOTE: —This is the eleventh of a series of articles on drugs by Professor Lewitus. The twelfth and concluding article of the series will appear in an early number.—EDITOR.

* * * *

GNAT (BLACK FLY) REPELLENTS

No. 1.—Castor oil	℥j
Pennyroyal	℥ij
Refined pine tar oil	℥iij

M. ft. lotio.

Sig. Smear the exposed parts of the body with this mixture.

No. 2—Fish oil soap	1 pound
Fresh cylinder oil	3 quarts
Water	1 gallon
Kerosene extract of pyrethrum	1 gallon

M. ft. emulsio.

Directions: The water and soap are heated to near boiling and then stirred vigorously while the mixture of kerosene extract and cylinder oil is slowly poured in.

Dysentery, White Scours or Infectious Diarrhea of Sheep

Often large sheepmen with the most expensive winter lambing barns have suffered the heaviest winter lambing losses from white scours infection. It is a trite saying that "the more money you put into a winter lambing shed, the less you will take out." Carefully analyzed, this indicates several things. Large lambing barns have been, in the past, built with little or no thought of letting sunlight into the barn. Sunlight is the greatest of germ destroyers and the greatest force that can be supplied the new born lamb. Even diffused light that passes through light clouds contains violet rays and is beneficial to the young. Ordinary window glass excludes the violet ray. Hence, the best way to utilize one of the large, poorly lighted sheep barns for safe winter lambing is to build some cheap sheds outside with either straw tramped in between double chicken wire, or rough lumber constructed sides and cover with a tarpaulin. The shed should open to the south and should be about the size that is built by many of the successful range winter lambers who use straw and sage brush, something like 15 feet deep, 6 feet high in front and 4 feet behind.

Unless the weather is stormy, the winter drop should take place outside and the mothering pens should be under the tarpaulin covered sheds. After four days, the ewe and the lamb can be taken inside the large barn for housing but the south end of the large building barn should be fitted with doors hinged at the top and when there is sunshine they should be lifted to let in the sun for the young lambs. The violet rays of the sun will pass through the tarpaulin covering the sheds unless it is badly soiled. Lambs that live past the fourth day seldom die of white scours; hence, the wisdom of keeping them outside. It is a known fact that two days of sunshine exposure on the part of a band of lambs badly white scours infected will stop the infection. Hence, as a preventive, cod liver oil, which carries both vitamin A and vitamin D by sunshine exposure, should be sprayed on grain hay if it is being used. Dosage of about a half an ounce per ewe three times a week will help fortify the unborn lamb against this vitamin A deficiency, which seems to predispose to white scours infection.

Crude cod liver oil may be purchased at approximately $19.00 per 30-gallon barrel, F.O.B. seaport. Several western range sheepmen who have suffered heavy losses and who are forced to feed grain hay which is deficient in vitamin A, are going to try this plan out. Alfalfa hay helps to some extent to correct vitamin A deficiency but sunlight seems to be the greatest panacea and cure-all. It is free and we should use it.

When white scours infection has once appeared in the lamb, the best treatment is dosing with the acidophilous cultured milk —a treatment developed by the Oregon State College, Veterinary Division. Sunlight is also helpful. By using the ultra-violet ray lamp, ailing lambs may be given the same stimulus they receive from sunshine.

Salem, Oregon W. H. LYTLE

Bacterin Dosage

As a rule, one can give from one-half to three-fourths the average bovine dosage to a full-grown sheep, and one-third the bovine dosage to a lamb.

In administering biologics to weak, "paper-skinned" lambs, evidently full of parasites, it is a good routine to give a small amount of bacterin or other biologics in one axillary space, and from 10 to 20cc calcium gluconate and dextrose, or a little camphorated oil, say 3 to 5cc, in the other.

Laxative for Sheep

In many cases, where there is stasis in the rumen and omasum, we first give 50cc calcium gluconate hypodermically in the axillary space. Then mix two ounces of aromatic cascara; one dram of fluid extract of nux vomica; four ounces molasses or corn syrup, or even strained honey, and one pint of mineral oil. Shake well and give four ounces twice daily. Sheep must be medicated very carefully, and not too much given at a time.

Canine Pediatrics

V. Carbohydrate-Fat Balance in Feeding Puppies*

By D. M. CAMPBELL, Chicago, Illinois

FATS and carbohydrates generally are regarded as the fuel or energy components of foods although proteins, too, contribute a share of calories.

Fats represent a highly concentrated energy source which may be stored within the body for future needs. They are additionally important as the normal carriers of the fat-soluble vitamins A and D.

Carbohydrates are the primary source of quickly usable heat and energy, but except for small reserves in the form of glycogen in the liver they cannot be stored unless in the processes of metabolism they are converted into fats.

IN the artificial feeding of very young puppies the selection of fats and carbohydrates is critical. Consideration must be given to the relative amounts of each and to the total caloric value of the carbohydrate-fat combination. Clinical data confirm the close theoretical relationship between improper carbohydrate-fat intake, either qualitative or quantitative, and disturbances of digestive processes involving pancreatic activity, rate of absorption, and blood sugar levels—disturbances which may be reflected throughout the animal's system.

It is generally realized by now that digestive idiosyncrasies of the young puppy proscribe excessive amounts of ordinary milk fat because of physical and chemical incompatibilities. The exact nature and cause of the incompatibility of cow's milk fat for very young puppies is not entirely understood; however, there seems to be no doubt that such incompatibility directly or indirectly affects and disturbs normal functioning of the pancreas. A broad and general analogy to the dietary regimen of the diabetic individual may be applied. Although diabetic affliction focuses primarily on carbohydrate metabolism, dietary treatment and control of the diabetic patient also involves a very strict regimentation of other dietary components, especially the fat.

FATS are compounds of glycerin and fatty acids. The particular nature of a fat is determined by the specific fatty acids in its composition. Certain of these differences are measurable. Thus, some fats are hard and contain greater proportions of the saturated fatty acids. The soft fats contain a substantially greater proportion of the glycerides of the unsaturated fatty acids. Among the established analytical standards for determining the various so-called fat constants is the iodine number which is an empirical means of determining the relative proportions of unsaturated fatty acid radicals in a fat. The unsaturated fatty acid molecule derives this popular designation from the fact that it combines, or is capable of combining, with other elements. Thus, the unsaturated fatty acid molecule is highly susceptible to oxidation, that is, under appropriate conditions it will combine with free oxygen of the atmosphere or with oxygen that may be derived from other substances. The manner in which this process functions leads to certain end products such as aldehydes and organic acids, the latter with characteristics somewhat different from the original fatty acid. Many of these products resulting from oxidation of unsaturated fatty acids are of a toxic or irritating character. Others have definite biological value for certain species. The significance, therefore, of the stability of fatty acids composing a given natural fat has many ramifications, hence the importance of the iodine number as a preliminary

* Prepared in collaboration with Dr. G. C. Supplee and J. H. LeWare, Bainbridge, N. Y.

item of consideration in determining the adaptability of a natural fat for a given purpose.

The iodine number of a fat is the number of grams of iodine 100 grams of the fat will absorb. The iodine number of natural bitch's milk fat is 58.3, while the iodine number of cow's milk fat is only 32. Human milk fat lies midway between with an iodine number of 43.

THE difference in fatty acid characteristics as measured by the iodine number of cow's milk and bitch's milk may be assumed to reflect the particular fat needs of the respective young of the two different species and has a teleological explanation in the fact that the body fats of herbivorous species contain more hard fats, derived from the saturated fatty acids, while the body fats of carnivorous species are made up of more soft fats, derived from the unsaturated fatty acids.

This difference is greatly emphasized when it is recalled that 9% of bitch's milk is fat, while only about 3.54% of cow's milk is fat. Thus it is apparent that if the gross fat content of cow's milk were increased from 3.54% to 9% by adding more cow's milk to match the gross fat content of bitch's milk, the relatively proportionate amount of the saturated fatty acid would be increased greatly and an undesirable nutritive condition would be worsened.

Although the saturated fats are less prone than the unsaturated fats to reaction with other substances and to combinations with individual elements, they are also less easily emulsified. The first step in the process of digestion and assimilation of a fat involves an emulsification with the digestive fluids. This purely physical phenomenon of emulsification, with concurrent exposure of the fat particle to the dispersed enzymes of the digestive fluids, facilitates splitting of the fat and its subsequent absorption. It is obvious, therefore, that a hard fat, or one more resistant to emulsification, other things being equal, is less easily digested, particularly by those species whose digestive systems are ordered to deal with softer fats.

THE importance of providing the suckling young of a species with the proper ratios of saturated and unsaturated fatty acids is well illustrated in artificial feeding of human infants. A vast body of clinical data shows that any excess of the saturated fatty acids of cow's milk in an artificial feeding formula for babies results in digestive and intestinal disturbances. For this reason most pediatricians modify cow's milk to reduce its fat constituent. This practice harmonizes with the fact that the iodine number of human milk fat is higher than that of cow's milk fat.

A probable explanation for the different biological effects of various milk fats in the feeding of the young of a species lies in the inherent differences in processes of digestion and absorption.

AN ingested fat is acted upon by the pancreatic fluids. Such pancreatic enzymes as lipase split the fat into its component fatty acids. The fatty acids are acted upon by the bile salts, which prepare them for absorption into the blood stream, either through emulsification or by rendering them water-absorbable. The young of any species will be able to utilize effectively those fats which are acted upon efficiently by the enzymes of its gastric juices. If an excess amount of fat is ingested, or if the fat is relatively indigestible, the normal pancreatic and bile fluids are unable to cope with it, and in an attempt to digest the incompatible or excessive fat, pancreatic and bile secretion will be overstimulated. This condition may become pathological and is reflected quickly in green and liquid feces and frequently develops into severe cases of enteritis and diarrhea. (See case histories.)

Aside from considering the specificity of fatty acid ratios, caloric values and quantitative and qualitative differences in fats as found in cow's milk and bitch's milk, it is necessary to consider again the phospholipides, sterols and related compounds.

TO the phospholipides are attributed functions in three metabolic processes; namely, the deposition of fat, cellular oxidation and the selective absorption and mechanism of the cell.

Natural fats, therefore, carry as an inherent part of their physical compositions, lipide matter other than the glycerides of the fatty acids. This lipide matter varies in composition and nature with the source and character of the natural fat. The functions of such lipide matter are of more importance in the mechanics of metabolism than the glycerides of the fatty acids which comprise the greater proportion of the natural fats, and serve to emphasize the significance of the wide distribution of the lipides in natural materials. In a previous article in this series reference was made to the fact that all natural proteins have associated or prosthetically bound with them lipide matter of varying characteristics. Cholesterol and certain varieties of phosphatides are almost universally found with natural protein, especially the protein of milk, and it is of further significance that these lipide matters are normally associated with the milk proteins to the maximum degree demanded for the proper nourishment of the young. It has been determined, for example, that the lactalbumins of milk have associated with them phospholipides, cholesterol and unidentified lipide matter entirely different in character from milk fat and the cholesterol content of such lipide material associated with the lactalbumin accounts for more of the total lipide content than does that found in the milk fat itself. The significance of lipides, other than the glycerides of the fatty acids, therefore, in digestion, assimilation, and regulation of tissue and cell functions, is greater than is obvious from consideration of mere gross quantitative fat content of a given food or dietary.

In considering the inter-species adaptability or incompatibility of milk, the character of the lipide matter carried by the fat components is of importance parallel to corresponding similar considerations regarding the protein components. Thus, it is logical to advance the hypothesis that the compatibility or incompatibility of proteins may be due largely, if not entirely, to the character of the lipide materials associated with them.

NATURE, and an impressive accumulation of clinical feeding data on the carbohydrate requirements of infants, point the way to selection of the optimum carbohydrate component of any simulated milk. Although the very young of various species can utilize other sugars, lactose has unique advantages as a caloric source, which are enhanced by its peculiar suitability to the carbohydrate function in maintaining blood sugar levels, which function in turn is regulated by the rate of hydrolysis and the comparative rate of absorption of the glucose and galactose resulting from such hydrolysis.

Lactose is hydrolyzed immediately upon entering the stomach, it has been shown, and part of the glucose thus formed may be absorbed immediately through the stomach, the duodenum, or the upper intestine. The degree to which glucose absorption occurs in the presence of galactose, which also results from the hydrolysis of lactose, apparently depends upon the blood sugar level at the time of ingestion. If the blood sugar level is low, comparatively high proportions of the glucose will be absorbed promptly; the galactose will be absorbed at a slower rate. If there is no immediate demand for replenishment of the blood sugar level, the absorption rates for both glucose and galactose will decrease. Since lactose cannot be absorbed without undergoing hydrolysis, and since evidence exists that the rate of hydrolysis of lactose may be determined by the blood sugar level, it is indicated strongly that, in contradistinction to other carbohydrates for which results of similar studies are available, lactose serves a function in maintaining an appropriate balance or equilibrium of the blood sugar level.

Since blood sugar levels reflect a correlation with pancreatic activity for the secretion of insulin, which in turn is a regulatory mechanism for maintaining blood sugar within proper limits, it is apparent that the balancing or regulating effect of lactose inherently involved in the phenomenon of slow absorption, must be interpreted as having a specific purpose in the nourishment of the young.

These general principles, however, do not imply that puppies, or any other animals, are endowed with unrestricted automatic capacity to regulate blood sugar levels by way of pancreatic activity beyond the scope characteristic of the species.

Certain other circumstances must be considered in determining proper levels of lactose for puppy nutrition. The hydrochloric acid content of the canine gastric juice is relatively high in comparison with that of other species which predicates a potentially high gastric capacity. Thus, ingestion of excessive amounts of lactose by a very young puppy would initiate a series of physiological excesses which might terminate in disaster for the pup. The reactions to inordinate lactose intake would follow in this sequence:

THE high gastric activity characteristic of the canine species would effect quick hydrolysis of the excessive lactose as soon as the latter entered the stomach, thereby making available for immediate absorption abnormal amounts of glucose, resulting in hyperglycemia. This in turn would stimulate excessive demand upon the pancreas for excretion of insulin into the blood stream. If prolonged, such excessive demand upon the pancreas for insulin would result in due course in a depletion or exhaustion of the pancreatic function, which if not corrected or if maintained in a routine dietary, obviously would lead to sustained hyperglycemia, a complete disbalancement of the blood stream, and consequent dysfunction of other physiological processes. Furthermore, the excessive demand upon the pancreas for insulin accompanying the hyperglycemic condition undoubtedly could result in an over-compensatory reaction of excessive insulin secretion into the bloodstream with too rapid or too complete exhaustion of glucose and consequent hypoglycemia.

These pathological phenomena do not apply to lactose alone. It is well known that dogs, and particularly young puppies, do not tolerate carbohydrates, especially the sugars. Lactose is cited only because recent evidence clearly indicates that certain conditions, which are accentuated highly in the gastric processes of puppies, lead to quick and excessive absorption of glucose. Excessive carbohydrate ingestion causes further unfavorable reactions in the young puppy. Excess hydrochloric acid, accompanying over-stimulated secretion of gastric juice, is beyond the normal neutralizing capacity of the duodenal area. Therefore, material of excess acidity is passed along into the intestine. The intestinal enzymes such as erepsin and trypsin cannot function in a hyperacid environment. This condition in turn leads to further digestive disturbances with common manifestations and obvious lack of assimilability. Of particular significance also is the fact that food emptied into the intestines, difficult to digest irrespective of the cause, in turn demands and stimulates abnormal pancreatic and bile secretions. These general conditions center especially upon the carbohydrate and fat components of the dietary. The "bilious attacks" experienced in the case of humans resulting from the so-called rich foods or fats incompatible or difficult of digestion are nothing more nor less than an excessive bile secretion stimulated by physiological efforts to complete the digestion as normally intended.

FAT and carbohydrate metabolism, therefore, must be considered jointly. The difficulty concerning fat and carbohydrate balance in the young puppy centers primarily in the pancreatic and bile secretions; dysfunction soon is reflected in the blood stream and throughout the body. Coma, convulsions, inanition, and manifestations of ravenous hunger on the part of very young pups are characteristic symptoms in the diagnosis and detection of fat and carbohydrate disbalancement.

Typical case histories showing the reaction of very young puppies to fat and carbohydrate intolerance, involving both incompatible fat and excessive carbohydrate, including lactose and other sugars, are presented herewith:

Case No. 43.—English setter; male; weight at birth, 310 gms.; suckling period, 3 days; then weaned to artificial formula with ratios of major components of:

Carbohydrate to fat	5.35:1
Carbohydrate to protein	2.74:1

Weight at time of weaning, 366 gms.; weight after 3 days on formula, 362 gms.

Feces: Changed from light yellow to yellow green.

On the 7th day of age the formula was again changed to one having ratios of:

Carbohydrate to fat.....3.41 : 1
Carbohydrate to protein.....1.96 : 1

Weight after 3 days on new formula, 367 gms.

Feces: Were maintained semi-formed, greenish-yellow in color.

On the 10th day of age the formula was again changed to one having ratios of:

Carbohydrate to fat.....0.60 : 1
Carbohydrate to protein.....0.27 : 1

Weight after one day on the changed formula, 308 gms.

The pup died on the second day following the last change in formula.

Feces: During the 24-hour period prior to death defecation was frequent; the feces being watery and green in color.

Post-mortem examination revealed telescoped intestines and enteritis.

Diagnosis: The highly incompatible formula provided immediately after weaning, particularly in reference to high fat and carbohydrate ratios, followed by frequent changes in dietary at short intervals, resulted not only in physiological but also in physical injury, causing death.

Case No. 9.—Cocker spaniel; female; weight at birth, 292 gms.; no suckling period.

Ratios of artificial formula:

Carbohydrate to fat.....1.04 : 1
Carbohydrate to protein.....0.70 : 1

Weight, after 7 days, was 276 gms.

Feces: Progressive change from meconium to light brown to watery green.

On the 8th day the formula was changed to one having ratios of:

Carbohydrate to fat.....3.41 : 1
Carbohydrate to protein.....1.96 : 1

Feces: Green watery progressing to dirty gray; voided at frequent intervals.

The post-mortem examination showed extensive enteritis.

Diagnosis: Incompatible food, particularly with reference to fat and carbohydrate; condition accentuated by further disbalancement of the new formula started on the 8th day was the cause of death.

Case No. 182.—Cocker spaniel; male; weight at birth, 223 gms.; suckling period before weaning to artificial formula, two days.

Ratios of artificial formula:

Carbohydrate to fat.....0.35 : 1
Carbohydrate to protein.....0.38 : 1

Weight after 25 days, 412 gms.

Feces: Formed light yellow progressing to dark brown. Normal.

The pup grew to maturity.

Diagnosis: The artificial formula was entirely compatible; the ratios of major components were similar to those of bitch's milk and the fat and carbohydrate were compatible in quality as well as quantity.

Case No. 8.—Cocker spaniel; male; weight at birth, 136 gms.; no suckling period.

Ratios of artificial formula:

Carbohydrate to fat.....1.07 : 1
Carbohydrate to protein.....0.71 : 1

Weight after 8 days, 166 gms.

Feces: Semi-formed; color, dark yellow.

On the 9th day the formula was changed to one having ratios of:

Carbohydrate to fat.....3.41 : 1
Carbohydrate to protein.....1.96 : 1

Feces: Immediate change concurrent with change in formula from dark yellow to light watery gray.

The pup died on the day following change in formula. Coma, inanition and spastic convulsions typified the last 24 hours of life.

Diagnosis: The original formula was not compatible as to ratios of fat and carbohydrate, nevertheless the pup was able to maintain existence for 8 days. Change to the new formula, less compatible as to quantity and quality of carbohydrate and fat, resulted in prompt death, preceded by coma and convulsive spasms.

Case No. 60.—Shepherd; male; weight at birth, 472 gms.; no suckling period.

Ratios of artificial formula:

Carbohydrate to fat.....0.90 : 1
Carbohydrate to protein.....0.68 : 1

The original formula supplemented with 4 to 6cc of 5% karo solution at feeding periods; the resultant ratios of the combined formula and karo supplement were:

Carbohydrate to fat.....1.67 : 1
Carbohydrate to protein.....1.08 : 1

Weight after 9 days, 263 gms.

Feces: Frequently voided; non-formed. Color, brown to yellow to green to watery gray.

Died on the 10th day.

Post-mortem findings: No pathological lesions and no evidence of acute enteritis.

Diagnosis: The original formula, while approaching that of bitch's milk in ratio of major components, was improperly balanced with reference to total carbohydrate due to the karo supplement.

Case No. 79.—Beagle; female; birth weight, 325 gms.; no suckling period.

Ratios of artificial formula:

Carbohydrate to fat.....0.99 : 1
Carbohydrate to protein.....0.59 : 1

Weight after 11 days, 372 gms.

Feces: Partially formed; light brown color, progressing to non-formed yellow with increasing frequency.

Twelfth day change in formula to ratios of:
Carbohydrate to fat.....0.83:1
Carbohydrate to protein.....0.63:1
Feces: Less frequent; progressing to formed yellow brown to normal brown; firm normal feces.
Weight after 16 days on new formula, 705 gms.
The pup grew to maturity.
Diagnosis: The original formula was not entirely incompatible, although ratios of gross components approach those of bitch's milk, character of the fat not suitable. Change to new formula with only slight change in ratios of components, but with more compatible fat, resulted in immediate improvement in fecal condition and increased growth response.

Case No. 81.—Shepherd; male; weight at birth, 464 gms.; no suckling period.
Ratios of artificial formula:
Carbohydrate to fat.....0.99:1
Carbohydrate to protein.....0.59:1
Feces: Progressing from meconium to brown to yellow to non-formed slate gray color.
On the 11th day the formula was changed to one having ratios of:
Carbohydrate to fat.....0.83:1
Carbohydrate to protein.....0.63:1
Feces: Less frequently voided; progressing from slate gray to formed greenish gray to light brown to normal dark brown formed boluses.
Weight after 16 days on new formula, 755 gms.
The pup grew to maturity.
Diagnosis: The original formula less compatible for this pup than for Case No. 79, although both formulas were the same, approaching the ratios of bitch's milk. Change to the new formula with only slight change in ratios of components but with a more compatible fat, resulted in immediate improvement in the fecal condition and increased growth response.

THE foregoing case histories demonstrate that aside from the physiological response of artificially fed puppies to compatible and incompatible fat and carbohydrate dietary constituents, probably the most significant data of practical value to the veterinarian in the practice of canine pediatrics are to be found in the observation of the feces. This subject will be treated in detail in a later article, but at this time it is pertinent to summarize the characteristic pathological pattern of fecal transition, both as to rate of change and character of change exemplified in the preceding case histories. The typical evolution of fecal conditions of puppies receiving formulas of intolerant carbohydrate and fat components is characterized first by change in color from dark brown to a lighter brown. Frequently bright yellow, semi-solid feces will appear. Often, however, the change in color from dark brown may go directly to a dark green cast. The greenish-brown color may prevail for a relatively short or long time, depending upon the degree of incompatibility of the food and the degree of ability of the individual to cope with the intolerable components. In many cases, however, the dark green, greenish-brown feces may be followed promptly by feces more definitely green with virtually no brown color. At this stage the fecal boluses are rarely formed. The frequency of defecation increases. A diarrheal or watery condition is usual. Under the particular conditions of incompatibility involved, pups seldom die during the period of passage of frequent green feces. The next transition in color is from a green to a grayish-green with the green color gradually disappearing and the feces becoming more gray. Finally, the change is to a dirty white or creamy, grayish-white cast. Experience has shown that with the particular incompatibility now under consideration, and with dogs voiding feces of this character, death follows in practically 100% of the cases. We shall mention, however, in a later article, a series of exceptions wherein this type of fecal condition was noted and the dogs were brought to recovery, but in those cases the incompatibility did not lie in the fat or carbohydrate.

AS a general rule abnormal feces of the character described are more acidic than are normal feces. However, the pH value of such pathological feces extends throughout a wide range and is not diagnostic *per se*, of the conditions which cause the abnormality.

A commonplace explanation for various diarrheal conditions in the suckling puppy is that the bitch's milk is "acid". This stereotyped explanation, or excuse for lack of knowledge of inherent physiological processes contributing to such a condition, has about as much validity or foundation as the abracadabra of jungle voodooism.

The acidic aspect of pathological fecal conditions of either the suckling or artifi-

cially fed puppy lies entirely within the puppy, and does not reflect any acidity of the bitch's milk, evidence obtained from feeding tests indicates. Under experimental conditions it was determined that a bitch's milk, normal in so far as pH value is concerned, but not in correct balance with reference to fat or its digestibility, or carbohydrate, or some other feature, in the case of suckling pups, or even an alkaline artificial milk in the case of bottle fed puppies, led to the pathological fecal condition which frequently is attributed to "acid milk".

The basis for associating acidity with the fecal condition under discussion lies in the sequence of events previously described. The gastric acidity of the puppy is excessive for the character of the food ingested. This may readily occur even with bitch's milk. The excess acidity passes from the stomach through the duodenum; the duodenal secretions are inadequate to neutralize the excess acidity; the resulting hyperacid gastric content automatically stimulates the pancreas and bile secretions; and an excessive creating further digestive disbalancement. amount of bile is poured into the intestines, The manifestation of green feces is obvious, visual evidence of excessive bile secretion. Furthermore, certain toxic or incompatible fatty acids may irritate the gall bladder and stimulate further the excessive bile secretion.

IT IS apparent, then, in the light of the theory of bile and pancreatic activity, and from clinical feeding tests on very young puppies, that the fat and carbohydrate constituents of an adequate simulated bitch's milk must be selected with utmost discrimination both as to the compatibility of each, individually, and as to the balance of both as an interacting whole.

THE responsibility of protecting artificially fed puppies from fat-carbohydrate imbalance, and assuring for them properly formulated feedings, is one that the veterinarian, in the practice of canine pediatrics, is best equipped to assume when guided by a full understanding of the physiological processes involved in fat-carbohydrate metabolism of young pups.

A VIEW OF THE EQUINE CLINIC OF THE E. I. V. A. AT MECHANICSVILLE

Two hundred sixty-two veterinarians, from Iowa, Illinois, Nebraska, Wisconsin, and Minnesota, attended the eighth annual veterinary clinic sponsored by the Eastern Iowa Veterinary Association at Mechanicsville, Ia., June 21st. Surgical operations and autopsies were performed, diagnoses made, and various treatments and tests were demonstrated. Displays included handy devices and apparatus, and an extensive histological and pathological collection was exhibited by Dr. G. A. Kay. Arrangements for the clinic were made by Drs. C. H. Banks, Frank M. Wilson, and Fred J. Crow.

Poisonous Plants of the South

In a very excellent bulletin, "Poisonous Plants of Alabama," by Cary, Miller and Johnstone, 57 toxic plants are listed, of which 28 have been known to poison sheep. They are as follows:

1. Sorghum
2. Poison darnel
3. Indian turnip
4. False garlic
5. Blue flag
6. Pokeweed
7. Corn cockle
8. Celery leaved crowfoot
9. Seshan
10. Flowering spurge
11. Water hemlock
12. Fool's parsley
13. Rose bay
14. Racemose fetter-bush
15. Yellow jessamine
16. Common nightshade
17. Jimson weed
18. Button bush
19. White snake root
20. Wild black berry
21. Cotton and cotton seed
22. Mountain laurel
23. Black locust
24. Castor oil bean
25. Death camas
26. *Caubentia longifolio*
27. Cockle burr
28. Water parsnip

Black Nightshade (*Solanum nigrum*)
—Charlotte M. King.

Water Hemlock *(Cicuta maculata)*
The spindle-shaped and lower stem, the leaves, flowers and fruit are shown three-eighths natural size; also a cross-section of seed X4. The water hemlock is an extremely poisonous plant—U. S. Dept. of Agr.

While practically every community in the country has its own peculiar weeds and plants, this list may be taken as fairly representative of the southern states.

We like to emphasize the fact that a correct diagnosis of plant poisoning is often very difficult, unless one is familiar with the locality. The experienced practitioner soon learns what to expect from the season of year, location of farm and the presence of common toxic plants.

He knows, when he starts out on a call to John Smith's place along the river bottom, that he may expect to find water parsnip or some other such plant growing in damp, marshy places. He also is aware that when he is called to Jim Jones' farm, up in the hill country, he may find plants common to a dry soil.

This knowledge is a stepping stone to successful diagnosis, for treatment of affected animals is secondary, especially when in a hopeless condition. If he can warn his client to move his animals to a new pasture, he has performed the service expected of him.

For this reason we have not gone into detail on the subject of poisonous plants, for it is too extensive to discuss in a short space. Pammel's "Manual of Poisonous Plants" contains 977 pages, and every practitioner doing sheep practice work will do well to keep this volume at hand. Then, if he learns to identify the poisonous plants that grow in his immediate locality, he will be doing all that his country expects of him.

Abstracts

Infectious and Metabolic Diseases

For practical purposes the constitutional diseases may be grouped into diseases of metabolism, and infectious diseases.[87] In the former, constitutional irregularities of certain metabolic processes play an important rôle; in the latter a kind of constitutional variation in resistance toward the causative agent is of practical importance. The following constitutional diseases are of economic importance: Softness of the horny hoof, eczematous conditions of the legs, rickets, spavin, galls, in horses; tuberculosis, contagious abortion, sterility, and rickets in cattle; growth disturbances, enzoötic bronchitis and pneumonia in pigs.

Chronic Catarrh in Horses

The mucous degeneration of the turbinated bones is in reality a chronic catarrhal inflammation, and not an ostitis fibrosa.[88] The question as to whether the disease starts in the turbinated bones, or in the nasal sinuses has not yet been settled. Due to secondary invaders the catarrh may develop into a purulent condition. Accessory causes are the complicated anatomical relations of the nasal sinuses, and disturbances in the development of the molars; there is a possibility that the closure of the aditus naso-maxillaris is inherited.

Roaring in Horses

The right vocal cord is always involved in the origin of the inspiratory sound,[89] even when the left vocal cord alone is paralyzed. This offers an explanation for the fact that the sound may be very loud, but that the lesions seen with the laryngoscope are nevertheless very slight. On the other hand, the lesions on the left side, as seen with the laryngoscope, may be very extensive, yet no abnormal sound is produced. The most common cause of roaring is strangles, especially when the retropharyngeal lymph nodes are diseased. The reason for this is that inflammatory edema and toxins produced by the streptococci are capable of passing to the recurrent nerve, causing degeneration of the nerve fibers. The left nerve is thinner and more sensitive than the right. Other causes of roaring are influenza, plumbism, coital exanthema, lathyrus poisoning, and traumatic injuries to the recurrent nerve. The prognosis is always doubtful, and usually bad. Definite proof that roaring is hereditary is lacking.

Causes of Sterility in Cattle

The therapy of sterility used to be dominated by bacteriology.[98] It is true, this branch of science cleared up certain details of this disease; nevertheless, it is now plain that the solution of the problem of sterility can be expected only by coöperation with parasitology, biology, and zoötechnics. The newer methods of obtaining spermatozoa and testing them systematically are of great aid to the practitioner in diagnosing the etiology of sterility in an entire herd. Morphological changes in the sperms, such as detachment of the head, curling of the tail, protoplasma-inclusions, and discolorations may be noted on microscopical examination. In young bulls the spermatozoa frequently possess a certain yellowish tinge. This is a rather desirable characteristic, as it indicates probably a high vitamin A or B_2 content; at any rate, it does not signify sparseness of semen. Loss of fertility is encountered often when cattle are fattened for market. It is not widely known that the artificial conditioning of exhibition cattle adversely affects their reproductive abilities. On the other hand, certain feeds, such as bran, green forage, and others, have a salutary influence upon fertility. Thus, a sterile bull, showing only a few immobile spermatozoa in its semen, has been restored to fertility completely by feeding bran, and by injecting wheat germ oil.

[87] Koch, W. Constitutional diseases. (Transl. title). *Deutsche Tierärztl. Wchnschr.* 45:781-783.

[88] Berge and Pallaske. Mucous degeneration of the turbinated bones in horses. (Transl. title). *Deutsche Tierärztl. Wchnschr.* 45:825-829.

[89] Wester, J. Roaring in horses. (Transl. title.) *Arch. Tierheilk.* 71:391-411.

[98] Siebenga, J. Sterility. (Transl. title.) *Tijdschr. v. Diergeneesk.* 64:689-693.

Purpura Hemorrhagica

Yatren,[90] an iodoform substitute of the formula $C_9H_4HSO_3IOHN$, has given excellent results in the treatment of morbus maculosus, especially when used in the early stages of the disease. The curative action is seen in the receding of the petechiæ as early as 24 hours after the first injection. The petechiæ, as well as the edema, disappear gradually during the course of the treatment. The exanthema, too, usually disappears after the infusion of the yatren solution. The colic symptoms, occasionally seen, vanish entirely. The pulse becomes stronger; the temperature falls to normal. The general appearance becomes visibly bettered. The technic of medication consists of intravenous infusion of a 4% solution of yatren; 10gm or 250cc of the 4% solution constitute the optimal daily dose. This amount may be given in a single injection. The infusion is to be repeated until improvement becomes manifest.

Electro-Thermal Treatment in Horses

The use of a Siemens ultra-thermo apparatus[91] with a wave length of six meters has given gratifying results in the treatment of horses. The therapeutic action of the ultra-short waves consists of the production of an active hyperemia in the depth of the tissue by means of the warmth created there. This hyperemia lasts for some time and cannot be counteracted by the application of adrenalin. Aside from this hyperemia a so-called stipple-heat is developed which is said to produce a certain curative action. The explanation for this is that small bodies absorb a larger amount of heat than the surrounding medium; thus, bacteria may be killed. Of course, proof for this statement does not exist, but the rapid healing of infected wounds and of furuncles points in this direction. Indications for ultra-short wave therapy are: arthritis, podotrochilitis, bursitis, tendinitis, and colics.

[90] Aehle. Treatment of petechial fever in horses. (Transl. title.) *Zeitschr. f. Veterinärkunde* 49:252-267.
[91] Driest, P. A. V. Short wave therapy for horses. (Transl. title.) *Zeitschr. f. Veterinärk.* 49:406-415.

Cystic Calculi in a Mare

In an eleven-year-old mare the bladder was observed to be distended. When the mare voided urine, the latter appeared inspissated, of the color of loam, and streaked with blood.[92] Palpation of the bladder disclosed the presence of calculi. With the aid of a metal catheter the calculi were extracted on two succeeding days. The calculi weighed 125gm; they consisted of calcium carbonate, ammonium urate, and traces of calcium oxalate. After treatment consisted of irrigation of the bladder with a 1% solution of rivanol, a 2% solution of boric acid and paraffin oil. Orally 20gm of hexamethylene tetramine were administered daily. Four weeks later the hexamethylene tetramine was superseded by 20cc of muriatic acid in the drinking water. Recovery was complete after a lapse of ten weeks.

Summer Sores in a Horse

A horse exhibited raw surfaces on the fetlock and fetlock joints of both hind legs. The affected areas were covered with numerous proliferations ranging in size from that of a bean to that of a walnut.[93] A 10% solution of wood tar in formaldehyde with a little alcohol added for better solubility, applied to these places, gave gratifying results. The mixture was applied first daily, later every second or third day. After about 45 days about two-thirds of the eczematous area was healed up. The remaining lesions, mostly around the fetlock, gave promise of rapid healing.

Oxygen Therapy

Meyer [94] describes four cases in which the medicinal therapy has been supplemented by oxygen therapy. The oxygen was applied by inhalation as well as by subcutaneous and intravenous injection. Inhalation was usually given in fractioned doses, comprising four to five inhalations, lasting from one to one and a half hours. In exceptional cases continuous inhalation

[92] Heizmann. Bladderstones in mares. (Transl. title.) *Zeitschr. f. Veterinärk.* 49:393-395.
[93] Krüger, H. Treatment of papilloma of the fetlock. (Transl. title.) *Zeitschr. f. Veterinärk.* 49:391-393.
[94] Meyer, H. Oxygen therapy in army veterinary practice. (Transl. title.) *Zeitschr. f. Veterinärk.* 49:322-336.

lasting from ¾ to 1 hour, was given. When oxygen was applied subcutaneously it took 9 to 21 hours before it was absorbed completely. The dose for subcutaneous application amounted to about 18 to 25 liters. The more grave the disease, the quicker is the oxygen absorbed. The intravenous injection requires great care on account of the danger from emboli. Combining the inhalation with the subcutaneous application is necessary to favorable results.

Shipping Diseases of Cattle

Cattle exposed to the virus of infectious bronchitis and bronchopneumonia begin to cough after an incubation period of four to seven days.[95] Sometimes, the fever rises very high. The eyelids, the upper respiratory organs, and the joints exhibit swellings; the udder is inflamed. Autopsy reveals marginal hepatization and dispersed pneumonic areas, ranging in size from a pea to a fist. Prevention is accomplished by proper care and hygiene during transportation; disinfection of market stables and auction stables; disinfection of the vehicles for transportation; disinfection of the clothing of the stable attendants. The therapy consists in the daily application of subcutaneous injections of iodine ether, phlebotomy, and fresh air.

Vitamin E Deficiency in Cattle

The common forage plants,[96] as a rule, contain sufficient amounts of vitamin E. However, cows being fed industrial by-products, often suffer from vitamin E deficiency when they are heavily in milk. Lately, even the natural forage, grass and hay, has become more or less deficient, due to the excessive use of artificial fertilizers. In both sexes vitamin E deficiency causes a certain form of sterility which is corrected easily by the administration of wheat germ oil, which is a rich source of vitamin E. Moussu claims that contagious abortion, retention of afterbirth, and certain calfhood diseases are due to lack of vitamin E. During the 5th to 7th months of gestation the growth of the fetus, the vitamin requirements of the mother, and the tendency to abortion, are at their maximum. If a vitamin E deficiency exists without the presence of abortion germs, then the fetus is carried to maturity, but the new-born calf is unable to resist bacterial infection. In severe cases of vitamin E deficiency the calves are weak, anemic, and emaciated, when born, and live only a few hours. In his fight against contagious abortion Moussu has used wheat germ oil with such excellent results in 700 cattle, that he came to the conclusion that contagious abortion does not occur unless there is a vitamin E deficiency present.

Causes of Retained Placentae

Mechanical hindrances, such as vaginal clamps, persistent hymen, premature closure of the cervix, and similar obstacles, rarely are the causes of retention of the afterbirth.[97] More frequently retention seems caused by a lack of expulsive power, due to failure of the womb to contract, or due to insufficient contraction. Often infection with *Brucella abortus* is encountered as the etiological agent. A most common form is idiopathic retention due to the diversion of large amounts of blood in connection with the beginning of lactation in high-producing milk cows. The most successful therapy is the manual extraction of the afterbirth. The disinfecting therapy is of minor importance.

In Denmark, a breeding circuit has been set up whereby a veterinarian examines all cows to be bred and in the afternoon secures semen from the sire to be kept for this purpose and inseminates the cows in the various herds. In 1936 more than a thousand cows were reported to have been bred by semen from one bull. Early this year a breeding circuit along this same line was organized in New Jersey, and in other states artificial insemination is being practiced.—HENRY WALLACE, Secretary, U. S. Dept. of Agriculture.

[95] Ellinger. Infectious bronchitis and bronchopneumonia in cattle. (Transl. title.) *Deutsche Tierärztl. Wchnschr.* 45:801-802.

[96] Hofstra, H. Vitamin E. (Transl. title.) *Tijdschr. v Diergeneesk.* 64:694-700.

[97] Liebsch, E. Retention of afterbirth in cattle. (Transl. title.) *Arch Tierheilk.* 71:1-40.